REST 3d TORTS-PH s 28
Restatement (Third) of Torts: Liab. Physical Harm § 28 (P.F.D. No. 1, 2005)

Restatement of the Law -- Torts

Restatement (Third) of Torts: Liability for Physical Harm (Proposed Final Draft) FNa
Current through April 2006

Chapter 5. Factual Cause

§ 28. Burden Of Proof

Proposed Final Draft No. 1
Case Citations July 2005 -- November 2005

Proposed Final Draft No. 1:

(a) Subject to Subsection (b), the plaintiff has the burden to prove that the defendant's tortious conduct was a factual cause of the plaintiff's physical harm.
(b) When the plaintiff sues all of multiple actors and proves that each engaged in tortious conduct that exposed the plaintiff to a risk of physical harm and that the tortious conduct of one or more of them caused the plaintiff's harm but the plaintiff cannot reasonably be expected to prove which actor caused the harm, the burden of proof, including both production and persuasion, on factual causation is shifted to the defendants.

Comment on Subsection (a):

a. Burden of proof of factual cause on plaintiff. Subsection (a) restates the general rule that the plaintiff has the burden of proof on factual causation, one of the elements of a prima facie case. Consistent with the burden of proof in civil actions, plaintiff must demonstrate by a preponderance of the evidence that a defendant's tortious conduct was a factual cause of harm. This burden consists of the two elements of the burden of proof: the burden of production, which requires the introduction of sufficient evidence to permit a rational factfinder to make a determination that a defendant's tortious conduct was a factual cause of the harm, and the burden of persuasion, which requires that the factfinder be persuaded from the evidence that factual causation more likely than not exists. Subsection (a) reflects no substantive change from Restatement Second of Torts § 433B(1), although it makes clear that the plaintiff's burden of proof relates to factual causation and omits the substantial-factor language from the Second Restatement.
The plaintiff must persuade the factfinder that factual cause exists by a preponderance of the evidence. Many courts employ a different standard when testimony is provided by medical or scientific experts, requiring the witness to testify to a "reasonable degree of medical [or

scientific] certainty." These courts seemingly defer to the standard of proof imposed by the medical or scientific communities. This deference is mistaken because the medical and scientific communities have no such "reasonable certainty" standard. Thus, the only meaning such a standard has to medical or scientific experts is that conjured by the individual expert or that provided by the attorney who hires the expert. The reasonable-certainty standard provides no assurance as to the quality of the expert's expertise, investigation, methodology, or reasoning. Rather than defer to a nonexistent and meaningless standard that is subject to manipulation by counsel or the expert witness, this Section adopts the same preponderance standard that is universally applied in civil cases. Direct and cross-examination can be employed to flesh out the degree of certainty with which an expert's opinion is held and to identify those opinions that are speculative and therefore inadmissible. FN*

b. Reasonable inference and speculation in proving causation. To isolate and determine whether an act was a factual cause of an outcome requires consideration of whether that outcome would have occurred without the act having taken place. As philosophers have taught, factual cause is not a phenomenon that can be seen or perceived; instead, it is an inference drawn from prior experience and some, often limited, understanding of the other causal factors--the causal mechanism--required for the outcome. Thus, all causal determinations require inferential reasoning. In some cases, the inference is quite powerful, as when a pilot, who failed to obtain an adequate briefing about the weather, flies into a storm and crashes, killing the passengers. In other cases, the inference may be quite weak, as when a person exposed to a suspected toxic substance develops a common form of cancer. Sometimes the inferences are submerged in the testimony of what appears to be a percipient witness, as for example when an eyewitness testifies that she observed the defendant crush the plaintiff's skull with a baseball bat. Sometimes the inferential reasoning required is quite evident, as when a child is found drowned in a pool that did not have a lifeguard present. Reasonable inferences are matters left to the jury's collective experience and common sense. But courts continue to regulate the cases that are submitted to juries by requiring that sufficient evidence be introduced to justify drawing a reasonable inference of factual causation. When the inferential leap from the evidence to the conclusion is too great, courts intervene to declare that juries may not speculate, and the matter is removed from the province of the jury. This procedure is not unique to factual causation, but exemplifies the procedural requirement of sufficient proof of each factual element of a claim.

The difficulty that courts confront is that the line between reasonable inference and prohibited speculation is one of the more indistinct lines that exists in law, and also is one on which reasonable minds can and do differ. Different courts draw those lines at different points at different times; comparison of cases is very difficult because modest differences in the evidence can substantially affect the power of an inference. Thus, it is not possible to state specific rules that locate the line between permissible inference and prohibited speculation.

Of course, plaintiff need not prove defendant's tortious conduct was a cause of the harm with a high degree of certainty. The civil burden of proof merely requires a preponderance of the evidence, and the existence of other, plausible causal sets that cannot be ruled out does not by itself preclude plaintiff from satisfying the burden of proof on causation. The difficulty is often that evidence does not provide any reasoned method for determining what the respective probabilities are for the potential causes. It is in cases of this genre that the general approach of a given jurisdiction toward the degree of freedom afforded juries to make a reasonable judgment in the face of uncertainty is critical.

In tort cases in which the defendant's tortious conduct is clear, many courts are lenient about the plaintiff's proof of causation, especially if the plaintiff has done all that is reasonably possible by way of gathering and presenting evidence of causation. Perhaps the best example of easing the plaintiff's burden on factual causation is found in those courts that have adopted a presumption of causation in negligence per se cases. Other courts have adopted a presumption of causation when the defendant fails to warn or provides an inadequate warning in products-liability cases. The use of presumptions in negligence per se and warnings cases is by no means universal, but it does reflect some courts' willingness to adapt the burden of proof depending on the type of tortious conduct and the difficulties of proof the plaintiff faces.

Beyond negligence per se and warnings cases, in unusual circumstances in addition to those in Subsection (b), courts may shift the burden of proof on causation to defendants. Courts have shifted the burden of proof on the aspect of causation that requires identification of the actor who committed the tortious conduct--there being adequate evidence that the tortious conduct caused the plaintiff's harm. These rare cases are characterized by a close relationship among the actors who potentially caused the other's harm, the actors having superior knowledge of the relevant circumstances, and the person harmed having no reasonable prospect for obtaining evidence of causation.

In some cases the specific facts of the defendant's tortious conduct and its relationship to the harm to the plaintiff may be sufficient to justify a reasonable inference of causation. Yet it is clear that the fact of the defendant's tortious conduct and harm to a plaintiff within the scope of the risk created by that conduct cannot alone be sufficient in all cases to permit an inference of causation. As a matter of scope of liability, defendant's tortious conduct must increase the risk of harm to the plaintiff. See § 29. If nothing further were required, other than adequate evidence of tortious conduct, plaintiff's burden of production on causation would always be satisfied. Thus, only when the tortious conduct reasonably could be found, after the fact, to have increased the risk of harm to a greater extent than the risk posed by all other potential causes would an inference from tortious conduct alone be permissible. Of course, this standard underscores the critical role of the identification of and elimination vel non of those other potential causes.

In the end, the line between permissible inference for the jury and impermissible speculation is one that must be determined based on the specific facts of the case and the jurisdiction's general procedural approach to the allocation of decisionmaking authority between judge and jury.

c. Toxic substances and disease

(1) Introduction. Cases involving toxic substances often pose difficult problems of proof of factual causation. These problems can also arise in cases involving activities that may cause disease, such as continued repetitive motion. Sometimes it is difficult to prove which defendant was connected to the toxic agent, see Comment *o*, or whether an adequate warning would have prevented the plaintiff's harm. See Comment *b*. The special problem in these cases, however, is proving the connection between a substance and development of a specific disease. In all of these cases, the requirement to prove factual causation remains the same; the plaintiff must prove it by a preponderance of the evidence, and the standards for factual causation set forth in §§ 26-27 continue to apply.

In most traumatic-injury cases, the plaintiff can prove the causal role of the defendant's tortious conduct by observation, based upon reasonable inferences drawn from everyday experience and a close temporal and spatial connection between that conduct and the harm. Often, no other potential causes of injury exist. When a passenger in an automobile collision suffers a broken

limb, potential causal explanations other than the collision are easily ruled out; common experience reveals that the forces generated in a serious automobile collision are capable of causing a fracture. By contrast, the causes of some diseases, especially those with significant latency periods, are generally much less well understood. Even known causes for certain diseases may explain only a fraction of the incidence of such diseases, with the remainder due to unknown causes. Causal agents are often identified in group (epidemiologic) studies that reveal an increase in disease incidence among a group exposed to the agent as compared to a group not exposed. Biological mechanisms for disease development--i.e., a series of causally linked physiological changes from exposure to disease developments--are frequently complicated and difficult to observe. Science continues to develop a better understanding of the biological steps in the development of diseases, but current knowledge in this respect is considerably more modest than for traumatic injury. As a consequence, courts in toxic-substances cases often must assess various alternative methods proffered with regard to factual causation.

Over the past several decades, courts have devoted a great deal of energy to the issue of causation in toxic-tort cases. Causation is a question of fact normally left to the jury, unless reasonable minds cannot differ. Appellate or trial-court review of jury findings affects the allocation of power between judges and juries. Until the early 1980s, a qualified expert witness's opinion that a toxic agent was a factual cause of the plaintiff's disease was treated as sufficient evidence. A few celebrated cases and case congregations, such as the Agent Orange and Bendectin litigations, led some courts to distrust juries' ability to resolve cases based on conflicting general expert-opinion evidence. Courts began to scrutinize the scientific evidence employed and to examine carefully the bases for an expert's opinion on factual causation. Some courts then tried to develop bright-line rules based on science for adequate proof of factual causation. The high water mark for this overreliance on scientific thresholds occurred in the Bendectin litigation when one court announced a blanket rule that a plaintiff could not make out a sufficient case without statistically significant epidemiologic evidence.

These courts may be relying on a view that "science" presents an "objective" method of establishing that, in all cases, reasonable minds cannot differ on the issue of factual causation. Such a view is incorrect. First, scientific standards for the sufficiency of evidence to establish a proposition may be inappropriate for the law, which itself must decide the minimum amount of evidence permitting a reasonable (and therefore permissible) inference as opposed to speculation that is not permitted. See Comment *b*. Second, scientists report that an evaluation of data and scientific evidence to determine whether an inference of causation is appropriate requires judgment and interpretation. Scientists are subject to their own value judgments and preexisting biases that may affect their view of a body of evidence. There are instances in which although one scientist or group of scientists comes to one conclusion about factual causation, they recognize that another group that comes to a contrary conclusion might still be "reasonable." These scientists' views reflect their scientific experience outside the courtroom. They may have different views about specific instances of conflicting scientific testimony in a courtroom. Judgments about causation may also be affected by the comparative costs of errors, as when caution counsels in favor of declaring an uncertain agent toxic because the potential harm it may cause if toxic is so much greater than the benefit foregone if it were not introduced. Courts, thus, should be cautious about adopting specific "scientific" principles, taken out of context, to formulate bright-line legal rules or conclude that reasonable minds cannot differ about factual causation.

This Comment is necessarily general. It addresses how methods of proof for traumatic injuries and for diseases may differ. Toxic-substance cases often involve statistical and group-based scientific studies that courts seldom confronted when the Restatement Second of Torts was published. Toxic agents and the diseases they cause differ, and methods of proof may vary accordingly. The law continues to evolve as courts are confronted with a variety of different circumstances related to different toxic substances, different disease, and the varieties of available evidence.

Scientific methods may advance in the future to better facilitate causation determinations for individuals, thereby obviating the need for statistically based group studies. While such techniques are largely unavailable today, dramatic advances in microbiology, genetics, and related fields have been made. These developments may produce new forms of evidence to which courts will adapt legal treatment of proof of causation.

Proof of causation often involves the admissibility of expert-witness opinions. Admissibility is governed by the law of evidence, and nothing in this Comment addresses that law. However, admissibility cannot be determined without reference to the substantive law. Moreover, courts may be required to examine scientific evidence when it is offered to prove agent-disease causation. That examination may occur either in the admissibility determination or in the determination whether the evidence is sufficient to meet the burden of production. These usually are separate issues and are subject to different legal standards. Courts, however, sometimes conflate these issues in the process of determining whether there is an adequate basis for an expert's opinion. When courts collapse the sufficiency determination into the question of the admissibility of an expert's testimony no subsequent inquiry into sufficiency is necessary, and the appropriate weight to give to an expert's opinion once it is deemed admissible is for the factfinder. The requirement of causation, the elements of agent-disease causation that are sometimes required when group studies are employed as proof, and the sufficiency of the evidence to meet the burden of production on causation are matters of substantive tort law, and they are addressed in the Restatement.

Most causation issues are resolved under the "but-for" standard for factual cause. See § 26. The plaintiff must prove by a preponderance of the evidence that, but for the defendant's tortious conduct with respect to the toxic substance, the plaintiff would not have suffered harm. When group-based statistical evidence is proffered in a case, this means that the substance must be capable of causing the disease ("general causation") and that the substance must have caused the plaintiff's disease ("specific causation"). In other cases, when group-based evidence is unavailable or inconclusive, and other forms of evidence are used, the general and specific causation issues may merge into a single inquiry. In any case, plaintiff's exposure to the toxic agent must be established.

Thus, courts often address "exposure," "general causation," and "specific causation." Nevertheless, these items are not "elements" of a plaintiff's cause of action, and in some cases may not require separate proof. So long as the plaintiff introduces admissible and sufficient evidence of factual causation the burden of production is satisfied. A court in a particular case may conclude that reasonable minds cannot differ about proof of factual causation under the general test *because* reasonable minds cannot differ on whether the plaintiff was exposed to the agent, whether the agent is generally capable of causing the disease, or whether the agent caused the plaintiff's disease in the specific case. These categories function as devices to organize a court's analysis, not as formal elements of the cause of action.

(2) Exposure to the agent. In evaluating factual causation, one issue that may arise is whether the plaintiff was exposed to the substance. Three primary means of exposure to toxic substances include inhalation, absorption, and ingestion, but others exist, such as injection or a fetus's transplacental exposure to agents in the mother's body. Often the method of exposure is critical to the type or extent of risk.

Exposure is frequently disputed in occupational-disease cases and hazardous-waste cases, while it is less often an issue in pharmaceutical cases. Proof of exposure may entail relatively straightforward historical facts, such as the presence of asbestos at the plaintiff's workplace or whether the plaintiff took a prescribed drug, or it may require complicated scientific evidence, such as dispersion modeling, to determine how and where the substance was transported. The latter form of evidence is often required in airborne-or groundwater-pollution cases. The intensity and duration of exposure (the "dose") affects the magnitude of the risks posed and the likelihood of causation.

(3) General causation. "General causation" exists when a substance is capable of causing a given disease. The concept developed because a prominent form of scientific methodology investigates causation on a group basis and therefore addresses whether an agent causes an increased incidence of disease in the group being studied. These studies proceed by comparing the incidence of disease in a group that has been exposed to the agent with the incidence of disease in a group of unexposed persons. The latter group's disease, thus, is attributable to causes other than the agent being studied. Traumatic-injury cases, by contrast, do not require this form of evidence because other causes that might explain the injury are absent, and we have a reasonably good understanding of the causal mechanisms involved from trauma to injury.

Occasionally, biological-mechanism evidence is sufficiently developed to prove general causation. More frequently, however, the evidence consists of scientific studies comparing the incidence of disease in groups of individuals (epidemiologic evidence) or animals (toxicologic evidence) with different levels of exposure. When a study finds a difference in the incidence of disease in the exposed and unexposed groups, an "association" exists between exposure and disease. Another type of epidemiologic study compares the extent of exposure among those with and without the disease. These studies seek to identify potentially causal substances at the aggregate population level--by finding a higher incidence of a disease in a group exposed to the substance (an "association").

Even when epidemiologic studies find an association between a substance and a disease, further analysis is necessary before a causal conclusion can be drawn. Scientists first systematically gather all of the studies that have been conducted and that are relevant to the causal question being investigated. When multiple studies exist, they are synthesized, either qualitatively in a review or quantitatively with a method known as meta-analysis. However, reasons may exist for disregarding or giving less weight to one or more of the available studies. If an association is found, epidemiologists use a number of factors (commonly known as the "Hill guidelines") for evaluating whether that association is causal or spurious. A spurious association may be the result of study errors--such as biases (scientists use "bias" to mean a source of error rather than as a predisposition to testify or decide a matter in an improper way) and uncorrected confounding factors (alternative causes that are responsible for the association, rather than the agent under study)--or sampling error (the result of small numbers of subjects and random chance). Similarly, a study may incorrectly fail to find an association that exists, because of study errors, especially when the disease is rare and an insufficient number of subjects exist to reveal any relationship.

Epidemiologists use statistical methods to estimate the range of error that sampling error could produce; assessing the existence and impact of biases and uncorrected confounding is usually qualitative.

Whether an inference of causation based on an association is appropriate is a matter of informed judgment, not scientific methodology, as is a judgment whether a study that finds no association is exonerative or inconclusive. No algorithm exists for applying the Hill guidelines to determine whether an association truly reflects a causal relationship or is spurious. Because the inferential process involves assessing multiple unranked factors, some of which may be more or less appropriate with regard to a specific causal assessment, judgment is required. For example, one of the Hill factors calls for an assessment of other scientific evidence that bears on the causal relationship under consideration. In some cases, there may be a substantial body of other evidence, while in other cases there may be little. The saliency of other evidence of causation often entails considerable judgment. Thus, in some cases, reasonable scientists can come to differing conclusions on whether a body of epidemiologic data justifies an inference of causation vel non. Similarly, reasonable scientists may, in some instances, disagree on whether the absence of an association is exonerative of the agent or is merely inconclusive.

Usually, other and unknown individual factors (causes) must concur with exposure to the agent for an individual to contract the disease. Group studies do not provide a basis for determining which individuals in a group suffer disease from exposure to the agent and which do not. More importantly, whenever other chemical, physical, or biological agents can produce the disease, group studies cannot distinguish which individual's disease was caused by exposure to a particular agent and which individual's disease was caused by another agent. So long as tort law adjudicates claims on an individual basis, specific causation requires attention even when general causation is established through the use of group studies.

Occasionally, courts have suggested or implied that a plaintiff cannot meet the burden of production on causation without epidemiologic evidence. Those cases often confronted a substantial body of epidemiologic evidence introduced by the defendant that tended to exonerate the agent as causal. Circumstances in individual cases, however, are sufficiently varied that almost all courts employ a more flexible approach to proof of causation--except in those cases with a substantial body of exonerative epidemiologic evidence. Epidemiologic studies are expensive and can take considerable time to design, conduct, and publish. For disease processes with long latency periods, valid studies cannot be performed until the disease has manifested itself. As a consequence, some plaintiffs may be forced to litigate long before epidemiologic research is available. Indeed, sometimes epidemiologic evidence is impossible to obtain, which may explain why neither the plaintiff nor the defendant is able to proffer supportive epidemiology. Thus, most courts have appropriately declined to impose a threshold requirement that a plaintiff always must prove causation with epidemiologic evidence, and, in some cases (as explained below), the evidence bearing on specific causation may be sufficient to pretermit the need to assess general causation.

(4) Specific causation. "Specific causation" exists when exposure to an agent caused a particular plaintiff's disease. Sometimes proof of specific causation is easy and collapses into proof of general causation, as when there are no alternative causal agents for a disease, and the disease is said to be a "signature" of the substance. In other cases, however, specific causation remains an issue even though general causation is established.

Scientists who conduct group studies do not examine specific causation in their research. No

scientific methodology exists for assessing specific causation for an individual based on group studies. Nevertheless, courts have reasoned from the preponderance-of-the-evidence standard to determine the sufficiency of scientific evidence on specific causation when group-based studies are involved. Properly understood and applied, this analytical framework provides a reasonable basis for determining specific causation in the absence of more particularistic evidence about the cause of plaintiff's disease.

Courts have reasoned that when a group study finds that exposure to the agent causes an incidence in the exposed group that is more than twice the incidence in the unexposed group (i.e., a relative risk greater than two) the probability that exposure to an agent caused a similarly situated individual's disease is 50 percent. Accordingly, courts generally hold when there is group-based evidence finding that exposure to an agent causes an incidence of disease in the exposed group that is more than twice the incidence in the unexposed group, the evidence is sufficient to satisfy the burden of production and permit submission of specific causation to a jury. In such a case, the factfinder may find that it is more likely than not that the substance caused the particular plaintiff's disease. The propriety of this "doubling" reasoning depends on group studies identifying a genuine causal relationship and a reasonably reliable measure of the increased risk. Courts appropriately have permitted expert witnesses to testify to specific causation based on the logic of the effect of a doubling of the risk and other considerations explained below that modify the probability of causation for a particular individual.

Additional considerations affect the propriety of determining the probability of specific causation based on the outcome of a group-based study. Depending on the state of the evidence about these additional matters, they may bear either on the sufficiency determination by the court or be relevant to the jury's determination. Thus, the extent to which the group-study outcome reflects the increased risk to the plaintiff depends on the plaintiff's similarity to those included in the group study. Relevant differences include whether: (a) the plaintiff was exposed to a comparable dose; (b) the plaintiff was not differentially exposed to other potential causes of the disease; and (c) the plaintiff has individual characteristics that might also bear on the risk of disease, such as age, gender, or general health, comparable to those in the study group.

The likelihood that an agent caused an individual's disease may be refined when there are independent, alternative known causes of the disease. The underlying premise is that each of these known causes is independently responsible for some proportion of the disease in a given population. Eliminating one or more of these as a possible cause for a specific plaintiff's disease increases the probability that the agent in question was responsible for that plaintiff's disease. Courts frequently refer to the elimination of other known causes for a plaintiff by employing the medical terminology of "differential diagnosis." The logic is sound, but the terminology and attribution are not. Assessing whether other causes can be ruled out (or in) as potential causes of a plaintiff's disease can provide probative evidence of specific causation. This technique is more accurately described as a "differential etiology." It is most useful when the causes of a substantial proportion of the disease are known. Then, the presence (or absence) of these causes for the specific plaintiff affects the probability that the agent in question caused the plaintiff's illness. When the causes of a disease are largely unknown, however, differential etiology is of little assistance. Evidence about biological mechanisms may also alter the likelihood that exposure to the substance caused plaintiff's disease, either by ruling out other known causes or by explaining why the suspected agent is a more likely cause of the disease than others.

For all of these reasons, any judicial requirement that plaintiffs must show a threshold increase in

risk or a doubling in incidence in a group study in order to satisfy the burden of proof of specific causation is usually inappropriate. So long as there is adequate evidence of general causation, courts should permit the parties to attempt to show, based on the sorts of evidence described above, whether the plaintiff's disease was more likely than not caused by the agent. Depending on the other factors detailed above, an increase of the incidence of disease less than a doubling may be sufficient to support a finding of causation, while in another case, even an increased incidence greater than two may not be sufficient. When the sufficiency of the evidence to meet the burden of production is at issue, courts should consider all of the evidence that bears on the matters discussed above and determine whether, in light of the general standard for sufficiency discussed in Comment *b*, the evidence would permit a reasonable jury to find that plaintiff's disease more probably than not was caused by exposure to the agent.

In most instances, differential etiology is not an appropriate technique for proving general causation. Nevertheless, in some limited circumstances courts have found that plaintiffs met their burden of proof of agent-disease causation without separate proof of general causation. Factors such as a good biological-mechanism explanation of how the agent could have caused the plaintiff's disease, a differential etiology ruling out other known causes, a reasonable explanation for the lack of general-causation evidence (and no contrary evidence of an absence of general causation), a short latency period and acute response, and the appropriate disease response to dechallenge (removal from exposure) and rechallenge (reexposure) to the agent, if combined and consistent, provide a persuasive basis for excusing the plaintiff from providing other proof of general causation.

(5) Multiple exposures and synergistic interactions. In some cases, a person may be exposed to two or more toxic agents, each of which is known to be capable of causing (general causation) the person's disease. The two agents may operate independently, in which case the incidence of disease in a group exposed to both will be additive--the excess incidence due to the first agent along with the excess incidence due to the second agent. Cases such as these present a relatively straightforward application of the principles set forth in Comment *c(4)*. If the toxic agents are attributable to the tortious conduct of separate actors, courts then face the question whether to apply the rule developed for multiple exposures in asbestos cases. This rule permits finding each actor's asbestos products to which the person was exposed to be a factual cause of the person's disease. See § 27, Comment *g*. Alternatively, courts might employ the traditional rule, requiring proof of which of the multiple exposures was a cause of the harm. At least where the biological mechanism by which disease develops is unknown, the asbestos rule is quite analogous and attractive as a means for adapting proof requirements to the available scientific knowledge. Apportionment of liability among those actors held liable is based on the comparative-responsibility rules in Restatement Third, Torts: Apportionment of Liability §§ 1-25. The alternative--the more traditional requirement of proof of which of the two toxic exposures was *the* cause of the disease--would require proof that does not exist, except on a probabilistic basis, as outlined in Comment *c(4)*.

Illustrations:

1. Abby was exposed to two different solvents while working in a laboratory. Each solvent contained a toxic chemical; one contained brion, and the other contained choron. After developing a disease, myeplopia, several years later, she sues the manufacturers of each

solvent, claiming that the manufacturers were negligent for including a toxic chemical in their solvents. Abby's evidence, presented by competent expert testimony based on valid scientific evidence, reveals that the increased risk of contracting myeplopia from the dose of brion to which she was exposed is insufficient to permit a finding of factual causation. Similarly, the increased risk of myeplopia from exposure to choron is insufficient to permit a finding of factual causation. However, Abby's evidence reveals that, while choron and brion operate independently (those exposed to both are only subject to an increased risk of the additive risks of each), the combined risk of contracting myeplopia due to exposure to both is sufficient to permit a finding of factual causation. Each of the manufacturers is subject to liability. See § 26, Comment *c*. Apportionment of liability between the manufacturers is governed by Restatement Third, Torts: Apportionment of Liability.

2. Same facts as Illustration 1, except that competent evidence shows that choron exposure increases the risk of myeplopia by 10 times, as does brion exposure. Competent evidence also reveals that the mechanism by which myeplopia develops is different for choron exposure and for brion exposure and that exposure to one or the other, but not both, is the most likely explanation for Abby's myeplopia. Abby cannot prove, however, whether choron or brion caused her myeplopia. Pursuant to § 28(b), the burden of proof on agent-disease causation is shifted to the manufacturers of choron and brion.

3. Same facts as Illustration 2, except that competent evidence reveals that choron and brion operate in precisely the same physiologic manner in the human body; they are interchangeable in their role in causing myeplopia. Exposure to each of choron and brion is a factual cause of Abby's myeplopia. See § 27, Comment *g*.

In some cases, as, for example, asbestos workers who smoke cigarettes, the two toxic agents together have a synergistic effect. This means that the excess incidence of disease among those exposed to both agents will be greater than the sum of the excess incidences found in those exposed to each separate agent. If the synergistic effect is sufficiently large, the excess incidence of disease due to the synergistic effect will be greater than the excess incidence due to each of the agents separately. In such circumstances, factfinders may infer that the combined exposure is a cause of the plaintiff's disease. This inferential process is similar to the one permitting a jury to find specific causation based on the increase in the incidence found from a general-causation study, such as those described in Comment *c(4)*. Although the reasoning for synergistic agents differs from that for nonsynergistic agents, the outcome is similar if the synergistic effect of the interacting agents is sufficiently large.

However, identification of both of the synergistic agents as a cause of the disease does not end the inquiry. Many causes exist for a given harm. See § 26, Comment *f*. Only those causes attributable to tortious conduct are legally relevant in determining liability and apportioning liability for plaintiff's harm. See Restatement Third, Torts: Apportionment of Liability § 26, Comment *m*. Thus, a natural condition, a genetic trait of the plaintiff, or a nonnegligent actor's conduct that are causes, in addition to a negligent actor's conduct, of the plaintiff's harm have no effect on the negligent actor's role as a cause of harm or on apportionment of liability. If more than one legally responsible agent is a cause of the plaintiff's harm, then apportionment of liability is based on comparative responsibility pursuant to Restatement Third, Torts: Apportionment of Liability §§ 1-25.

Illustrations:

4. Brett was occupationally exposed to asbestos for several decades. He also smoked cigarettes during approximately the same time period. Brett, who has developed lung cancer, sues Rossman, Inc., the manufacturer and supplier of the asbestos to which he was exposed, claiming that Rossman failed adequately to warn of the dangers of asbestos exposure. Brett provides competent expert testimony that, based on valid scientific studies, the dose of asbestos to which he was exposed increases the risk of contracting lung cancer by a factor of five (500%). The dose of cigarette smoke to which he was exposed increases the risk of lung cancer by a factor of 12 (1200%). However, the combined exposure to both asbestos and cigarette smoke increases the risk of lung cancer by a factor of 60 (6000%). Brett's evidence is sufficient to permit the factfinder to find that exposure to both asbestos and cigarette smoke were causes of his lung cancer. Because neither Brett nor Rossman claim that the smoking implicates tortious conduct, no apportionment of liability for Brett's lung cancer would occur, if the factfinder found in Brett's favor against Rossman.

5. Same facts as Illustration 4, except that Rossman successfully persuades the factfinder that Brett's smoking constituted negligence on his part. Neither Brett nor Rossman alleges any tortious conduct by the cigarette manufacturers. Liability for Brett's lung cancer would be apportioned between Brett and Rossman based on comparative responsibility according to Restatement Third, Torts: Apportionment of Liability § 7.

d. Burden of proof on magnitude of divisible harm. Harm may be causally divisible or indivisible. Indivisible harm may be either theoretically indivisible or practically indivisible. A harm is theoretically indivisible when each relevant actor's tortious conduct (including the injured person's contributory negligence) is a cause of the entire harm. In such a case, the only basis for apportioning the harm is comparative responsibility, not factual causation. See Restatement Third, Torts: Apportionment of Liability § 26. The burden of proof on the magnitude of harm when it is indivisible is on the plaintiff.

The type of injury is irrelevant to whether it is theoretically indivisible for apportionment-of-liability purposes. Death as an injury may not be divisible, but damages for death are divisible. Thus, for apportionment purposes, death may be divisible if the decedent was injured by the tortious conduct of one actor before dying, while the tortious conduct of another actor caused the death. When the law permits consideration of a hypothetical injury that would have occurred if subsequent tortious conduct had not occurred, in determining the extent of liability of the subsequent tortfeasor, the later injury is theoretically divisible. Thus, if a passenger in a negligently driven automobile is thrown out of the vehicle in an accident due to a defective door lock, latch, and seat belt, the automobile manufacturer may only be held liable for the harm that occurred diminished by the harm that would have occurred if the passenger had not been thrown from the vehicle in the accident, if there is adequate proof of the latter.

Harm is causally divisible when any party's tortious conduct caused less than the entirety of the harm for which the plaintiff seeks to recover. When harm is divisible, it should be apportioned based on causation, so that a party is held liable only for the harm factually caused by that party. See Restatement Third, Torts: Apportionment of Liability § 26, Comment *a*.

This Comment addresses the allocation of the burden of proof when the evidence reveals that the plaintiff's harm is theoretically divisible but there is doubt about the magnitude of harm caused by any party. If the plaintiff has the burden of proof on this issue and fails to introduce sufficient evidence of the magnitude of harm caused by a particular party, that party is not subject to liability. But if the burden of proof is shifted to the defendant under principles explained later in

this Comment and the defendant fails to introduce sufficient evidence to apportion the harm, then the defendant is liable for the entirety of the harm, which is then practically indivisible.

(1) Apportioning harm caused by the legally culpable conduct of multiple parties. When two or more actors (perhaps including the plaintiff) contribute to causing the harm, courts may confront the question of how much harm each actor caused. Often, the evidence will readily reveal that each actor caused some specific portion of the harm. Thus, if two actors each shoot negligently and one hits the victim in the eye, and the other hits the victim in the leg, the first actor is liable for damages flowing from the injury to the eye, and the second actor is liable for the damages flowing from the injury to the leg. Both actors are liable for damages that result from the combined injuries, such as occupational training necessitated by the combination of blindness and physical impairment to the leg. In other cases, all actors may have been a cause of the entirety of the harm, as when two cars driven negligently collide and a wheel from one car is thrown forcefully into a pedestrian nearby. Nevertheless, there are cases in which the evidence introduced by the plaintiff does not readily reveal how much of the harm was caused by each defendant's tortious act, although the evidence reveals that each such defendant contributed to causing at least some harm to the plaintiff. A modern example is "crashworthiness" cases in which the plaintiff, involved in an automobile accident caused by one tortfeasor, suffers enhanced injury due to a defect in the automobile that makes it uncrashworthy. In this and other cases, the magnitude of harm resulting from the initial accident and the magnitude of the enhanced harm caused by the uncrashworthy condition of the automobile may be uncertain. Historically, courts required plaintiffs to prove with a high degree of specificity the magnitude of the damages each defendant caused. This often resulted in plaintiffs' being unable to meet their burden of proof. The unfairness of this rule led several courts either to relax the quantity or quality of evidence of magnitude required or to hold that, when a plaintiff could show that each defendant caused some of the damages, each was jointly and severally liable for the entire damages unless the defendants could prove the magnitude each caused. Although the Restatement Second of Torts never made explicit this relaxation of the burden of proof, it is implicit in § 433B(2), which placed the burden of proof on apportioning harm suffered by the plaintiff on defendants. No need for proof permitting causal apportionment of the plaintiff's harm would be necessary if plaintiff initially bore that burden as an element of the prima facie case. Similarly, § 16 of Restatement Third, Torts: Products Liability, addresses the situation in which the plaintiff establishes increased harm because of a product defect, but proof does not permit determining its magnitude. The Products Liability Restatement imposes liability on the product manufacturer for the entirety of the harm. Thus, the Products Liability Restatement does not require the plaintiff to prove the magnitude of harm caused by each tortfeasor in a crashworthiness case and imposes the burden of proof on the party seeking to limit its liability on the ground that it caused less than all of the plaintiff's harm.

Consistent with both the Restatement Second of Torts and the Products Liability Restatement, this Comment provides that plaintiff need not prove the precise magnitude of harm caused by each defendant in order to make out a prima facie case: So long as the plaintiff proves that each defendant's tortious conduct caused some harm, the plaintiff has satisfied the burden of proof on factual cause. Conversely, a defendant who establishes that plaintiff's contributory negligence caused some of the plaintiff's harm need not prove the precise magnitude of damages attributable to that component of harm. This reciprocal treatment of plaintiff negligence is consistent with the overall approach of Restatement Third, Torts: Apportionment of Liability. The question of the

burden of proof when a party seeks apportionment of liability based on causation is addressed in Restatement Third, Torts: Apportionment of Liability § 26, Comment *h*.

(2) Apportioning harm between tortious conduct and innocent causes or nonparty actors. By contrast with Comment *d(1)*, causal apportionment of a plaintiff's harm may be required when a plaintiff had a preexisting symptomatic condition and the extent of enhanced injury is uncertain. Thus, a defendant whose tortious conduct aggravates plaintiff's preexisting back problem and causes greater pain is liable only for the harm caused by the aggravation and not for any harm due to the original condition. Such apportionment may also be required when the plaintiff suffers enhanced injury before trial for which the defendant is not liable, and the enhanced injury is not caused by tortious conduct or, if it is, the responsible actor is not a defendant in the suit.

The preexisting condition (or enhanced harm) may be the result of entirely innocent forces, as when a plaintiff is born with a congenital susceptibility to harm or develops such a susceptibility later in life. The condition may be the result of a lifestyle choice of the plaintiff, such as obesity from overeating or emphysema from cigarette smoking, or even the result of contributory negligence by the plaintiff. The condition may be the result of prior tortious conduct by a nonparty. The plaintiff may have already sued for and recovered damages for the preexisting condition from a nonparty.

Given the variety of circumstances and the predictable schism among the courts about how to allocate the burden of proof, the Institute takes no position on who should bear the burden of proof. The equities for imposing this burden on the plaintiff recede as: 1) the relative seriousness of the preexisting condition compared to the aggravation is small; 2) the preexisting condition was the product of innocent causes; 3) even when the preexisting condition was not the product of an innocent cause, the plaintiff has been unable to recover from anyone else for the preexisting condition; 4) the plaintiff was not contributorily negligent in causing the aggravation; and 5) the plaintiff is in no better position to prove the extent of aggravation than is the defendant.

Regardless of how the burden of proof is allocated, the preferred approach is to employ a modest threshold for the party with the burden of proof to satisfy the burden of production on the magnitude of harm. So long as there is some modicum of evidence that would permit the factfinder to make a causal apportionment, that course is preferable to making whichever party bears the burden of proof bear the entirety of the loss.

Comment on Subsection (b):

e. Burden shifting when plaintiff cannot reasonably demonstrate which of several tortfeasors caused the harm. Subsection (b) is based on the well-known case of Summers v. Tice and its adoption in § 433B(3) of the Restatement Second of Torts. This burden-shifting rule is commonly known as "alternative liability." The Second Restatement included a Comment raising a number of concerns about the scope of § 433B(3) that might be confronted in future cases, including possibly requiring joinder of all defendants, simultaneity of the tortious conduct by each actor, and the similarity of both the tortious conduct and the concomitant risks to the other. See Restatement Second, Torts § 433B, Comment *h*. Since the publication of the Second Restatement in 1965, courts have generally accepted the alternative-liability principle of § 433B(3), while fleshing out its limits. The advent of asbestos litigation and identification-of-defendant problems associated with those cases have produced a substantial number of alternative-liability claims. The inappropriateness of alternative liability for asbestos cases has

become well-established, see Comments *i* and *l*, but not without some fits and starts as courts sought the appropriate boundaries and reasoning for this outcome. In the end, courts have resolved exposures to multiple defendants' asbestos products in the different fashion described in Comment *l*.

f. Rationale. The rationale for shifting the burden of proof to defendants whose tortious conduct exposed the plaintiff to a risk of harm is that, as between two culpable defendants and an innocent plaintiff, it is preferable to put the risk of error on the culpable defendants. See Restatement Second, Torts § 433B(3), Comment *f*. In at least some cases, it appears that the defendants' better access to proof and doubts about the plaintiff's ability to extract that evidence from the defendants, even with modern discovery, have influenced the courts to employ burden shifting. Conversely, when plaintiff had a reasonable opportunity to identify the person whose tortious conduct caused the harm and negligently or undiligently failed to pursue that opportunity, courts have been disinclined to permit a plaintiff to invoke alternative liability.

The justification for alternative liability might logically begin with consideration of the single-defendant case in which plaintiff can prove tortious conduct but is unable to prove factual causation. Yet courts do not ordinarily shift the burden of proof in those cases. The explanation lies in the flexibility afforded in the standard for the burden of production on factual causation explained in Comment *b*. This flexibility enables courts to submit a case to a jury when plaintiff has made a plausible, if ambiguous and circumstantial, case for causation. That flexibility does not exist when there are multiple defendants, only one of which could have caused the plaintiff's harm, and the plaintiff has no evidence tending to show that a specific defendant's tortious conduct was a cause of plaintiff's harm. In these circumstances, no reasonable inference could be drawn that *more likely than not* any one of the defendants' tortious conduct was a cause of plaintiff's harm. Thus, the flexibility the single-tortfeasor situation affords courts, see Comment *b*, is often not available with multiple defendants. Alternative liability developed in response to this multiple-defendant situation.

g. Joinder of all defendants. Courts have insisted that all persons whose tortious acts exposed the plaintiff to a risk of harm be joined as defendants as a condition for alternative liability. When fewer than all such persons are joined in the case, the person who actually caused plaintiff's harm may escape liability. In addition, when fewer than all such persons are sued, a much weaker case exists for joint and several liability in those jurisdictions that retain it.

In the DES litigation, the predominant type of case in which plaintiffs have not joined all defendants, a better method exists for apportioning liability among defendants than contribution based on comparative responsibility. Thus, courts that have been willing to relax the plaintiff's burden of proof in DES cases when fewer than all DES manufacturers are joined have fashioned a modestly different scheme that modifies joint and several liability and adopts some form of market share to apportion liability among defendants. See Comment *o*.

The requirement of joinder of all persons does not prevent the plaintiff from settling with one or more of those persons either before or after filing suit. Similarly, although courts have not yet confronted the situation, it would be reasonable to excuse the plaintiff from this joinder requirement when an immunity or lack of jurisdiction prevents the joinder.

h. Each defendant acted tortiously. Unless all of the actors who may have harmed the plaintiff acted tortiously, the rationale for invoking alternative liability is absent. Courts continue, without exception, to turn away plaintiffs who are unable to establish this element.

Illustration:

6. Reed, a pedestrian, was injured by a sofa that was negligently or intentionally thrown from an upper-story hotel room during the celebration of an NCAA basketball championship. Reed sues all of the occupants of the 47 rooms from which the sofa might have been thrown. Reed must prove which of the defendants was responsible for throwing the sofa; the burden shifting provided in this subsection is unavailable to Reed in his suit because he has not shown that the occupants of each of the 47 rooms acted tortiously.
The result would be the same if two sofas fell simultaneously, one thrown negligently or intentionally from one room and the other not due to negligence of the occupant of the other room, and Reed, not knowing from which room the sofa that fell on him came from, sued the occupants of both rooms. Each of the possible causes of harm must be tortious for this subsection to be invoked.

i. Exposing plaintiff to the risk of harm. In Summers v. Tice, the connection between the defendants' tortious conduct and the plaintiff's harm was quite tight, regardless of the actual causal relationship. Both defendants were physically present, fired their guns in the direction of the plaintiff, and by doing so created an imminent risk to the plaintiff. Similarly, in many alternative-liability cases, a close connection exists between the risk of harm created by the defendants' tortious conduct and the harm suffered by the plaintiff. As that connection becomes more tenuous, even while each defendant remains a *possible* cause of the plaintiff's harm, courts generally refuse to invoke the rule in this subsection. Thus, for example, when a defective product injures a person who cannot identify which of several manufacturers made the specific product, courts refuse to invoke alternative liability against all manufacturers of the product. Similarly, courts refuse to permit plaintiffs in toxic-substances litigation to invoke the rule in this subsection. The unstated rationale in these cases seems to be concern over the administrative costs of imposing the burden of exculpation on a number of defendants who are only remotely implicated in the plaintiff's harm and for whom proving exculpation might be possible but burdensome. In at least some cases, but by no means all, courts are influenced by a lack of diligence by the plaintiff in obtaining or preserving evidence that would have permitted identification of the actor who caused the plaintiff's harm.

Illustrations:

7. Ken was hit by a taxi late one night after a substantial snowstorm. The skid marks left by the taxi revealed that after the driver slammed on the brakes and began skidding, the brakes failed. A nearby witness confirmed that the driver of the taxi appeared to be pumping the brakes furiously while the vehicle continued to roll. Because of the hour and lack of light, neither Ken nor the witness could identify the company name of the taxi. There are five different taxicab companies that operate in the community; each has a single cab. Examination of the five cabs reveals that each one has the same brake problem, caused by negligent maintenance, which permits the brakes to fail when a substantial amount of snow accumulates around them. The burden of proof on which taxicab company's vehicle ran into Ken remains with Ken because the other taxicab companies' negligence was too far removed to have exposed Ken to a risk of harm.
8. Five taxicabs were driven on city streets crowded with automobiles and pedestrians. The

five taxi drivers, operating independently, were negligently weaving in and out of traffic on the same street at the same time, narrowly averting collisions among themselves and with others. One of the taxicabs nicked Ken, a pedestrian, breaking his leg, while swerving to avert a collision with another vehicle. In the confusion, no witness could identify the taxi that hit Ken. The burden of proof on which taxicab company's vehicle hit Ken is shifted to the taxicab companies, if Ken joins all of them in his suit.

j. Multiple actors and nonsimultaneous tortious conduct. Subsection (b) is applicable when there are two or more actors whose tortious conduct expose another to risk. This is consistent with Restatement Second of Torts § 433B(3), which explicitly refers to the conduct of "two or more actors." There is a stronger intuitive appeal to alternative liability when there are only two defendants, and each is equally likely to have been the factual cause of another's harm, thereby leaving a plaintiff just a bit short of the evidence required to prove that one was the cause of harm by a preponderance of the evidence. Nevertheless, the rationale for alternative liability--shifting the burden of proof to the culpable parties who exposed an innocent person to risk--applies as well when there are more than two such culpable parties. Courts, since the Second Restatement, continue to apply alternative liability to those cases in which there are more than two tortious defendants.

So long as the actors' tortious conduct exposed the other to a risk of harm and the other requisites of this subsection are met, the actors' tortious conduct need not have occurred at the same time, nor need the tortious conduct of each be the same. Although simultaneous tortious conduct (or nearly so) existed in many of the early alternative-liability cases, no good reason exists for requiring simultaneity as a condition for shifting the burden of proof. Requiring simultaneous conduct may serve as a rough screen for having a sufficiently close connection between the tortious conduct and the harm, or it may serve imperfectly to limit the application of alternative liability to cases in which actors have no better access to proof than the victim. The former requirement is explicit in this subsection, and the latter is not required for invocation of alternative liability.

Illustration:

9. Lucia, while strolling through a heavily forested park, wandered out of the park and on to privately owned land, on which Chris was hunting. At the same time, Chris negligently fired his gun in Lucia's direction, Nicholas, a child of eight, who had the day before been negligently entrusted with a rifle by his father, Rich, also fired in Lucia's direction. By remarkable coincidence, a spring gun tortiously set the prior week by Allison, who owned the land on which Lucia wandered, was set off and fired a bullet in the direction of Lucia. Lucia was struck by one of the bullets and sues Chris, Nicholas, Rich, and Allison. Available ballistics evidence does not permit determination of the source of the bullet that harmed Lucia. Each of Chris, Nicholas, Rich, and Allison has the burden of proof to show that his or her negligence did not cause Lucia's harm.

k. One or more defendants' tortious conduct caused plaintiff's harm. Alternative liability is limited to those cases in which fewer than all defendants caused plaintiff's harm, and the plaintiff is unable to prove which one(s). When multiple actors are each a cause of all of another's harm, all such actors are liable for that harm; the form of that liability and apportionment among them is based on Restatement Third, Torts: Apportionment of Liability §§ 1-25. When multiple actors

are each a cause of a portion of another's harm, there may be problems of proof about how much harm each defendant caused. The problem is similar to the one addressed by alternative liability, but is dealt with in Comment *d*.

Illustration:

10. Phil, a passenger in an automobile passing through an intersection, was injured when first Elizabeth, and then, several seconds later, Joshua, driving in opposite directions on the intersecting road, went through a red light, and each collided with the car in which Phil was a passenger. After the collisions, Phil had a severely fractured hip, which required several reconstructive surgeries. In Phil's suit against Joshua and Elizabeth, uncontradicted evidence shows that Joshua would have collided with Phil's car, because of its slow speed, in virtually identical fashion, whether or not Elizabeth had first hit Phil's car. The evidence, however, is conflicting on whether Phil's harm occurred due to the first collision alone or the second collision alone, and on whether its severity was a consequence of both collisions. The jury should be instructed that, if it can find that one or the other of the collisions alone was the factual cause of Phil's harm, the verdict should be against the defendant responsible for that collision. If the jury finds that the severity of the fracture was a result of both collisions, both defendants would be liable for the entirety of the harm, pursuant to Comment *d*, unless one was able to show a basis for causal apportionment. Finally, if the jury finds that Phil's harm was caused either by Joshua or Elizabeth, but not by both, and cannot determine which, Subsection (b) shifts the burden of proof on causation to Joshua and Elizabeth. Unless one or both has introduced sufficient evidence to satisfy this burden, both are liable for Phil's harm.

In some alternative-liability cases, different actors play different roles with regard to the causal set that produces the harm. Thus, one actor's tortious conduct is a causal element in the set resulting in another's harm. When there is uncertainty about which of several other actors' tortious conduct provided another and different causal element in the set, the burden shifting provided in Subsection (b) is, nevertheless, appropriate.

Illustration:

11. Paula, a construction worker employed by a door and window subcontractor, was caulking the outside of windows and doors on a multi-story building under construction. Working from inside the building, she was required to lean out the window and apply caulking where necessary. Although required to do so by an applicable safety regulation, Able, the general contractor, did not provide safety straps for use by construction workers who were working on upper floors where a risk of falling existed. While Paula was leaning out an opening for a fire escape and caulking the outside, she was bumped by someone moving a piece of structural steel, which caused her to fall. Only two other workers were on the floor at the time of Paula's fall, David and Diana. Paula sues Able, David, and Diana. At trial, Walter, a security guard, testifies that he observed only David and Diana on the floor from which Paula fell both just before and just after her fall. Walter observed both being careless in their moving structural steel around the floor on which Paula was working. Although Able's negligence was a cause of Paula's harm, Paula may invoke Subsection (b) to shift the burden of proof to David and Diana on which one of them was moving the structural

steel that was also a cause of her fall and concomitant harm.

l. Exposure to multiple defendants' toxic products. As explained in Comment *e*, a significant number of asbestos cases have confronted the application of alternative liability. These cases are characterized by plaintiff's exposure to asbestos products of multiple actors, plaintiff's development of asbestotic disease decades later, and, often, plaintiff's inability to identify the asbestos products to which plaintiff has been exposed. As explained in Comment *i*, courts have refused to employ alternative liability to relax the requirement that plaintiff identify the defendant responsible for the product(s) that caused harm. When the plaintiff is able to identify multiple defendants, the rule in this subsection is inapplicable because all of the asbestos products to which plaintiff was exposed contributed to the harm, rather than a single defendant's product having caused the harm. Section 27, Comment *g*, addresses this situation and may be applicable, but asbestos cases nicely demark the distinction between this subsection and § 27, Comment *g*.

m. Procedural implications. Plaintiff must establish each of the requirements contained in Subsection (b) before the burden of proof on causation shifts to defendants. Thus, plaintiff must persuade the factfinder that each of two or more defendants engaged in tortious conduct that exposed the plaintiff to a risk of harm. As to the requirement that one or more of the defendants was a factual cause of the plaintiff's harm, the plaintiff may seek to prove which defendant caused the harm or that all defendants caused harm, as suggested in Illustration 10. If the plaintiff is successful in that effort, no burden shifting is necessary. If the plaintiff is not successful in that effort, the burden shift provided in this subsection applies so long as the factfinder is persuaded that the plaintiff reasonably is unable to prove which of the defendants caused the harm. The evidence of the tortious conduct and of the defendants' connection to that conduct ordinarily will support the further inference that the plaintiff cannot reasonably prove which defendant(s) caused the harm. A defendant who claims that the plaintiff reasonably could have done more must, as a practical matter, point to aspects of the plaintiff's evidence or present new evidence revealing some deficiency in the plaintiff's efforts to identify the defendant(s) who committed the tortious conduct.

Because the shift in the burden of proof depends on a finding that one or more defendants was a cause of plaintiff's harm and the plaintiff is reasonably unable to prove which one, a contingent instruction to the jury may be required. First, the instruction would provide that the jury must find for the defendants if it finds that there was no tortious conduct or that none of the defendants committed the tortious conduct. If plaintiff's evidence is sufficient to permit the jury to find that one or more defendants' tortious conduct was a cause of plaintiff's harm, the jury would be instructed to first decide whether the evidence persuades it of which defendant's(s') tortious conduct was a cause of plaintiff's harm. If the jury can determine which defendant(s) caused the harm, the matter of causation is resolved, and no burden shift is necessary. If the jury finds that one or more defendants' tortious conduct caused the harm, but cannot determine which one(s), the jury would be instructed on the shifted burden of proof imposed on the defendants.

Once plaintiff establishes the conditions for shifting the burden of proof, both the burden of production and of persuasion are shifted to defendants on the question of which defendant's(s') tortious conduct caused plaintiff's harm. Unless one or more defendants produces sufficient evidence to create an issue of fact either that another defendant's tortious conduct was the cause of plaintiff's harm or that that defendant's tortious conduct was not a cause of the plaintiff's harm, the court should rule for the plaintiff on this question as a matter of law. In addition, each of the defendants has the burden of persuading the factfinder either that that defendant was not a cause

of plaintiff's harm or that another defendant was the only cause of plaintiff's harm. Were that not the case, when one of two defendants caused the harm, as in Summers v. Tice, defendants who merely presented evidence that each was equally likely to have caused plaintiff's harm would avoid liability. Plaintiff retains the burden of proof on all other prima facie elements of the case. When multiple defendants are held liable pursuant to this subsection, whether liability is several (and therefore apportioned according to comparative responsibility), joint and several, or some hybrid depends on the law in the applicable jurisdiction. See Restatement Third, Torts: Apportionment of Liability § 17.

Illustration:

12. Alan, Barbara, and Chad were playing paintball together in a secluded portion of a forest. Before beginning each game, one of the three was assigned to scout the area for anyone else who might wander into the area and thereby be put at risk, while the other two competed. The person watching was to give an all-clear signal before each game began and then keep a watch at the only means of entrance and egress. While Alan and Barbara were playing, Penelope wandered into the area and was hit in the eye by a paintball fired by either Alan or Barbara, both of whom were firing paintballs simultaneously. Penelope sues Alan and Barbara claiming that each of them fired negligently after Chad had issued a warning that they should cease playing. Penelope also sues Chad, alleging in the alternative that he negligently failed to issue a warning of Penelope's presence. At trial, both Alan and Barbara testify that Chad issued no warning, and Chad testifies that he did. Penelope has satisfied her burden of production that each of the defendants acted negligently and that one of them caused Penelope's harm. The court should instruct the jury to decide whether Chad issued a warning of Penelope's presence. If the jury finds that Chad did not issue a warning, Penelope has established that Chad's negligence caused her harm (and that Alan and Barbara were not negligent in the manner alleged). If the jury finds that Chad did warn Alan and Barbara and that they negligently fired after the warning, each of Alan and Barbara bear the burden of demonstrating that his or her negligence did not cause Penelope's harm and that, unless one of them succeeds in persuading the jury of that fact, both should be found to be a factual cause of Penelope's harm.

n. Plaintiff negligence. Alternative liability was developed when plaintiff's own negligence constituted a complete bar to recovery. The adoption of comparative responsibility raises the issue of whether alternative liability can be invoked by a plaintiff who is also at fault. Because of the absence of any significant case law addressing this situation, the Institute takes no position on this issue.

o. Market-share liability. In a narrow range of cases, courts have been confronted with plaintiffs who have similar proof problems to those who seek to invoke alternative liability. These cases involve exposure to a toxic substance and the plaintiff's understandable inability to prove which manufacturer sold the product, often many years or decades before any disease becomes clinically evident. The prototype for this kind of case is the drug diethylstilbestrol (DES), a drug prescribed in the middle part of the 20th century to prevent miscarriage. DES, because it was not patented, was manufactured by hundreds of pharmaceutical companies. DES caused disease in the offspring of the mothers who took the drug, typically 20 years after exposure, and at a time when evidence of which manufacturer produced the DES the mother had consumed was quite

often unavailable. As explained in Comment *g*, courts refuse to employ alternative liability to permit these plaintiffs to shift the burden of proof to defendant-manufacturers.

A number of courts nevertheless adopted a new "market share" theory that permitted apportionment of liability among defendant-manufacturers based on each one's share of the relevant market for DES. Many of the details of the specific market-share theory adopted vary from court to court, but common to all is that liability is several, rather than joint and several, and is limited to the market share of each defendant, so that in theory each will pay roughly the amount that represents the overall harm caused by that defendant's DES. A roughly equal number of courts have declined to craft a new theory for DES plaintiffs, expressing concern that to do so would rend too great a chasm in the tort-law requirement of factual causation. Despite several decades of development, the number of jurisdictions that have addressed and resolved this question for DES victims is quite small; the vast majority of states has not yet been confronted with or decided this issue. However, with DES having been withdrawn from the market in 1971, a latency period of approximately 20 years, and very little judicial activity over the past decade, it appears unlikely that there will be any significant further development of market-share liability in the DES context. Virtually all courts that have considered the question have declined to apply a market-share liability theory to products that are not fungible and therefore do not pose equivalent risks to all of those exposed to the products.

Market-share liability, when the product is fungible and therefore poses equivalent risks, is attractive both from a compensatory and deterrence standpoint. If plaintiffs can demonstrate that the marketing and sale of a product was tortious and that the product caused their harm, they have a strong claim for compensation. More importantly, deterrence is better served by making manufacturers of toxic substances responsible for harm caused by defective products, even when the passage of time makes identification of the connection between manufacturer and plaintiff impossible. Using market share to determine each manufacturer's several liability provides an appropriate deterrent to tortious conduct because it imposes liability for approximately the amount of harm caused by each manufacturer's defective product. Market-share liability is more difficult to justify from a corrective-justice standpoint, but at least some commentators find corrective justice compatible with liability for imposing risk.

Even when serving compensation and deterrence goals, there are two difficulties with market-share liability. First, the administrative costs of determining each defendant's market share have been distressingly disproportionate to the compensation provided. Part of the reason was the natural intuition of courts to seek the local market most particular to the plaintiff, thereby increasing the likelihood that the manufacturer that provided the drug that harmed the plaintiff is among those held liable and facilitating the opportunity for defendants who did not provide the drug that harmed the plaintiff to exculpate themselves. Narrowly defined markets, however, require relitigation of the appropriate market in nearly every case. Later courts, appreciating this experience, employed broader, even national, markets to keep administrative costs within reason. Even then, concern about the existence and accuracy of market-share data is another aspect of administrative costs.

A second concern that deterred some courts from adopting a market-share theory is institutional capacity. Crafting a coherent market-share scheme that both relaxes the traditional tort requirement of factual causation and provides a workable market-share system is much more the type of lawmaking traditionally and appropriately a matter for legislative action than for common-law decisionmaking.

When market-share liability is limited to fungible products that pose equivalent risks to users who have no reasonable means to prove which manufacturer provided the product that caused plaintiff's harm, it has an exceedingly limited reach. New drugs are patented and therefore sold exclusively by a single manufacturer for a number of years after FDA approval-- DES was a notable exception. Only products that cause harm after a lengthy latency period between exposure and development of harm are likely to create the systemic proof problems that market-share liability addresses. Many toxic substances, including asbestos products, do not pose equivalent risks to all exposed to the products. And, when the defect in the product is a manufacturing defect, market-share liability would be unavailable unless each manufacturer's production and quality-control processes were so similar that the probability of a manufacturing defect was the same for each. While in theory a risk-adjusted market-share liability system might be attractive, the administrative costs imposed even by a pure market-share system augur against such efforts, and there is virtually no case support for a risk-adjusted market-share theory. Even among jurisdictions accepting a pure market-share theory, it has very rarely been applied outside of DES cases.

Because of the nearly even split among jurisdictions and the lack of an emerging consensus or trend, Restatement Third, Torts: Products Liability § 15, Comment *c*, explained the important considerations in crafting a market-share system and left the matter to the developing law. Since publication of the Products Liability Restatement, there have been no developments that would justify the Institute changing course. The lack of activity in this area may reflect the declining significance of the issue, as explained above.

REPORTERS' NOTE

Comment a. Burden of proof of factual cause on plaintiff. The "reasonable certainty" standard remains in wide, if uncritical, use. A number of courts, however, that have scrutinized the matter and recognized that the "reasonable certainty" standard is not necessary to screen out speculative opinions and may be detrimental to that goal have moved away from requiring that physicians or scientists utter the "reasonable certainty" incantation as a condition for the admissibility of their opinions. See Schulz v. Celotex Corp., 942 F.2d 204, 208 (3d Cir.1991) (holding that expert held opinion with requisite degree of confidence and therefore lower court erred in excluding testimony because expert neglected to state it was held with reasonable medical certainty); Poertner v. Swearingen, 695 F.2d 435, 438 (10th Cir.1982) (medical expert may employ preponderance of the evidence with regard to strength of opinion); Romano v. Atkins, F. Supp. 2d , 2004 WL 945140 (E.D.Pa.2004) ("What is important is not any particular phrase that the expert used, but that the opinion not be based on speculation or a mere possibility."); Estate of Patterson v. Fulton-DeKalb Hosp. Auth., 505 S.E.2d 232, 234 (Ga.Ct.App.1998) ("Thus, 'reasonable medical probability' has no greater meaning than a preponderance of the evidence, and the standard of proof is preponderance of the evidence as to medical causation."); Nunez v. Wilson, 507 P.2d 329, 332 (Kan.1973) ("[E]vidence of probative value should not be excluded from the jury's consideration merely because a medical expert cannot state a fact with absolute certainty."); Kramer v. EBI Cos., 878 P.2d 266 (Mont.1994) (preponderance of the evidence is standard required for medical testimony; testimony of a "possibility" does not meet that standard); Dallas v. Burlington N., Inc., 689 P.2d 273, 277 (Mont.1984) (observing that reasonable medical certainty is a concept "not well understood by the medical profession" and

that the goal of assuring medical testimony is not based on mere possibility is satisfied if "medical testimony is based upon an opinion that it is 'more likely than not' "); Shahan v. Hilker, 488 N.W.2d 577 (Neb.1992) (medical testimony is admissible if it is "sufficiently definite and certain that a conclusion can be drawn;" expert testimony "need not be couched in the magic words reasonable degree of medical certainty"); Kunnanz v. Edge, 515 N.W.2d 167, 173 (N.D.1994) ("Hypertechnical words are not necessary for the admission of an expert medical opinion; the test for admissibility is whether the expert's testimony demonstrates the expert is expressing a medical opinion that is more probable, or more likely than not."); Shumaker v. Oliver B. Cannon & Sons, Inc., 504 N.E.2d 44, 46 (Ohio 1986) ("It is well-settled that the establishment of proximate cause through medical expert testimony must be by probability. At a minimum, the trier of fact must be provided with evidence that the injury was more likely than not caused by defendant's negligence."); Dellenbach v. Robinson, 642 N.E.2d 638 (Ohio Ct.App.1993) (trial court erred in excluding testimony of expert who did not understand what "reasonable medical certainty" meant but who would provide an opinion based on more likely than not); Welsh v. Bulger, 698 A.2d 581, 585- 586 (Pa.1997) ("We do not, however, require experts to use 'magic words' when expressing their opinions.... Instead, we look at the substance of their testimony."); Stormo v. Strong, 469 N.W.2d 816, 824 (S.D.1991) ("[T]he test is only whether the expert's words demonstrate that he or she was expressing an expert medical opinion."). For a recent judicial recognition of the infirmity of continued use of "reasonable degree of medical certainty," see Bara v. Clarksville Mem. Health Sys., Inc., 104 S.W.3d 1 (Tenn.Ct.App.2002) (commenting on the ambiguity, excessive rigorousness, and lack of meaning of "reasonable degree of medical certainty" and therefore returning to the preponderance standard in all civil cases); Drexler v. All Am. Life & Cas. Co., 241 N.W.2d 401 (Wis.1976) ("No particular words of art are necessary to express the degree of medical certainty required to remove an expert opinion from the realm of mere possibility or conjecture. The test to be applied is whether a reasonable interpretation of the expert's words demonstrate that he was expressing his expert medical opinion."); Weber v. McCoy, 950 P.2d 548, 551 (Wyo.1998) ("Wyoming does not require that an expert use the magic words 'reasonable medical probability' in order for his opinion to be considered a competent medical opinion."). Rules 702 and 703 of the Federal Rules of Evidence, which govern the admissibility of expert-witness testimony and require that experts employ reliable methodology and reasoning in forming their opinions, do not impose a requirement that an expert testify that an opinion is held with a reasonable degree of scientific or medical certainty. See In re Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir.1994); United States v. Cyphers, 553 F.2d 1064, 1072 (7th Cir.1977). But cf. Grant v. Farnsworth, 869 F.2d 1149, 1152 (8th Cir.1989) (chiropractor who could only "guess" as to the causal relationship between harm and tortious conduct and who could not state his opinion with reasonable certainty was properly excluded from testifying as an expert). On the issue of whether state law or the federal rules of evidence govern in cases in federal court when state law provides the governing substantive law, compare In re Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir.1994) (holding that because the standard for admissibility of expert testimony is, in reality, about the plaintiff's burden of proof, state law governs despite conflicting federal rules of evidence) with United States v. Cyphers, 553 F.2d 1064, 1072 (7th Cir.1977) (holding that Rule 702 is the sole standard applicable to determine admissibility of expert testimony). See also Robin K. Craig, *When* Daubert *Gets* Erie*: Medical Certainty and Medical Expert Testimony in Federal Court*, 77 DEN. U. L. REV. 69 (1999).

When the issue of whether a plaintiff will develop additional injury in the future arises, courts routinely permit expert testimony that is expressed in terms of "probable," "likely," or similar indications of certainty. See Phillip E. Hassman, *Admissibility of Expert Medical Testimony as to Future Consequences of Injury as Affected by Expression in Terms of Probability or Possibility*, 75 A.L.R.3d 9, 19-20 (1977) ("When the testimony is couched in such terms, it would seem that the expert considers the probability of their occurrence as being better than 0.5, and such testimony has almost always been permitted."); Dalebout v. Union Pac. R.R., 980 P.2d 1194, 1198-1199 (Utah Ct.App.1999). Despite the ALR author's sanguinity about the meaning of terms such as "probable" or "likely" to experts, more precise characterizations such as "more likely than not" or "greater than 50 percent probability" are less likely to be misunderstood.

The justification for use of the reasonable degree of medical certainty standard is to avoid testimony based only a possibility or a guess. See Hohnstein v. W.C. Frank, 468 N.W.2d 597, 603 (Neb.1991) ("[The] rule flows from the requirement that a decision cannot be based upon guess or speculation); Drexler v. All Am. Life & Cas. Co., 241 N.W.2d 401, 432 (Wis.1976). The preponderance standard serves that function by eliminating testimony that is based on probabilities of 50 percent or less. Equivalent expressions that the expert is not testifying merely of a possibility or speculating also serve the goal of ensuring that experts have a firm belief in the correctness of their opinions. One such standard is found in the testimony of Benjamin Peirce in the celebrated 19th-century trial of Robinson v. Mandell. Peirce testified with regard to whether the signature on a will was forged by tracing it from a genuine specimen: "Under a solemn sense of the responsibility involved in the assertion, I declare [that the signature was forged]." See LOUIS MENAND, THE METAPHYSICAL CLUB 173 (2001). Any of these standards serve the goal of avoiding speculation better than the "reasonable certainty" standard because they employ a vocabulary that has a commonly understood meaning.

For a stinging critique of the use of "reasonable medical [or scientific] certainty," along with an inquiry into its genesis and development, see Jeff L. Lewin, *The Genesis and Evolution of Legal Uncertainty about "Reasonable Medical Certainty,"* 57 MD. L. REV. 380 (1998) (tracing history of phrase to local usage early in the 20th century in Chicago to accommodate Illinois law that barred speculative opinions, subsequent adoption in a single decision in Illinois, and widespread propagation in other jurisdictions "through unreflective imitation of models provided in a best-selling manual on trial technique"). For commentators who have decried the lack of meaning of this phrase among physicians and other scientific experts, see Edward R. Stein, *The Direct Examination of the Expert Witness*, *in* FAUST F. ROSSI, EXPERT WITNESSES 193, 220 (1991) ("[P]ractically nobody knows what it means. Many experts can be frightened off the witness stand if they have not been prepared for a question that includes the word "certainty." They think it means 'for sure,' a concept that does not exist for most expert witnesses."); Mark D. Howard, *Proving Causation with Expert Opinion: How Much Certainty Is Enough?*, 74 ILL. B.J. 580, 584 (1986) ("Most experts, other than professional witnesses, are unfamiliar with the 'reasonable certainty' language used in court."); Jonas R. Rappeport, *Reasonable Medical Certainty*, 13 BULL. AM. ACAD. PSYCHIATRY & L. 5, 5 (1985) ("Doctors testify to it daily, but they do not know what it means."). Indeed, those providing advice for medical experts explain that the phrase is a "legal fiction." HAROLD A. LIEBENSON, YOU, THE MEDICAL WITNESS 129 (1961) ("What is this thing called 'reasonable degree of medical certainty'? It is a legal fiction."). For other commentators critical of the "reasonable medical certainty" standard, see Bert Black, *A Unified Theory of Scientific Evidence*, 56 FORDHAM L. REV. 595, 669

(1988) ("[R]easonable certainty adds nothing to the law except the opportunity for confusion."); James E. Hullverson, Jr., *Reasonable Degree of Medical Certainty: A Tort et a Travers*, 31 ST. LOUIS U. L.J. 577, 577 (1987) ("The phrase has been a source of confusion, frustration, and endless interpretation for litigants, trial judges, and appellate courts.").

One of the Reporters had an opportunity to discuss this issue with several scientists and a physician at a meeting held on January 27, 2005 of the Committee on Alternate Models to *Daubert* Standards of the Science, Technology, and Law Program of the National Academy of Sciences. All agreed that "reasonable degree of medical [scientific] certainty" is not a term that is employed in their disciplines or that has any specific meaning to them. One scientist could not construct a meaning for the term, and one other suggested it might mean absolute certainty, but then recognized that the "reasonable" modifier was inconsistent with that interpretation. Several suggested that they could not interpret the extent of certainty the term implied in the abstract, as they are accustomed to adjusting the degree of certainty based on the comparative costs of a wrong decision. Thus, a physician may employ a benign treatment for a life-threatening illness even though the physician thinks it highly unlikely that the treatment will be successful. The law has already decided that, while it attempts to minimize errors, for those that do occur the law is indifferent to errors favoring plaintiffs or defendants in civil cases and adopted a preponderance standard that reflects that determination. That legal principle should be conveyed to expert witnesses, and their testimony should meet that standard.

While the standard for admissibility and sufficiency of the evidence to meet the burden of proof are technically distinct, the standard for admissibility is less stringent than the standard for sufficiency. Thus, it is illogical to employ a higher standard for admissibility than for the burden of producing sufficient evidence. For proof of causation in toxic-substances cases, however, courts have collapsed the admissibility and sufficiency standards for expert testimony about causation. See Reporters' Note to Comment *c(1)*. Regardless of whether these standards are treated as the same or not, there is no basis for imposing a standard requiring that an opinion be held to a higher degree of certainty than a preponderance of the evidence. Cf. In re Paoli R.R. Yard PCB Litig., 35 F.3d 717 (3d Cir.1994) (concluding that Pennsylvania's rule requiring an expert to testify to reasoanable degree of certainty is not just about admissibility of evidence, which would be governed by contrary federal procedural law contained in the Federal Rules of Evidence, but is about the burden of proof imposed on a plaintiff and therefore governed by state law).

Comment b. Reasonable inference and speculation in proving causation. On the necessity for inferential reasoning to determine causation, see Glanville Williams, *Causation in the Law*, [1961] CAMBRIDGE L.J. 62, 70 n.22 ("[I]n one sense, hypothesis and speculation are essential for determining causal connection, since every statement of causal connection asserts what would have happened if the facts had been different."); H.L.A. HART & A.M. HONORE, CAUSATION IN THE LAW Lxi (2d ed. 1985) (counterfactual inquiry always required for causal assessment).

The Harper, James, and Gray treatise observes that in determining what inferences of causation are permissible, one finds "the same test of delusive exactness that is generally used to gauge the legitimacy of the basis for an inference from circumstantial evidence." 4 FOWLER V. HARPER, FLEMING JAMES, JR. & OSCAR S. GRAY, THE LAW OF TORTS § 20.2, at 93 (2d ed. 1986). Not surprisingly, therefore, one finds quite variable and, indeed, inconsistent results. For inconsistent decisions on virtually identical facts from the same court, compare E. Tex. Theatres,

Inc. v. Rutledge, 453 S.W.2d 466, 468-470 (Tex.1970) (reversing lower courts that had permitted submission of plaintiff's case claiming that negligence of movie theatre in failing to make efforts to control rowdy crowd caused plaintiff's harm) with Marek v. S. Enter., 99 S.W.2d 594, 596-597 (Tex.1936) (contra). Sometimes even the same judge appears to have been inconsistent. Compare Wolf v. Kaufman, 237 N.Y.S. 550 (N.Y.Sup.Ct.App.Div.1929) (Finch, J.) (holding jury question on causation was not created by evidence that decedent fell on unlit stairs because "it would be solely a conjecture for a jury to draw the conclusion that the deceased fell down the stairs because of the absence of light" due to defendant's negligence) with Ingersoll v. Liberty Bank of Buffalo, 14 N.E.2d 828 (N.Y.1938) (Finch, J.) (holding that it was for jury to decide whether, on the one hand, decedent fell down stairs due to a heart attack or dizziness while carrying 32-pound box or whether, on the other, decedent fell on second-to-last step, which was found broken and had been negligently maintained by defendant, and injuries from fall caused his death).

For citations to cases taking a lenient view of plaintiff's burden on causation, see 4 FOWLER V. HARPER, FLEMING JAMES, JR. & OSCAR S. GRAY, THE LAW OF TORTS § 20.2, at 94-101 (2d ed. 1986); see also DAN B. DOBBS, THE LAW OF TORTS § 173, at 421 (2000) ("Courts are avowedly liberal with such causation issues and many cases have permitted an inference of causation along these lines."); Wex S. Malone, *Ruminations on Cause-in-Fact*, 9 STAN. L. REV. 60, 94-97 (1956) (describing liberality courts have permitted juries in finding factual cause through the substantial-factor test but also providing copious citations to courts using a more rigorous standard of proof).

Professor Malone's insight into the rigor with which courts hold plaintiffs to their burden of proof on factual cause is that it depends on the importance of the claim being enforced and the connection between the harm and the interests that the legal claim seeks to protect. Id. at 72-73. This insight may help explain at least some cases in which the plaintiff claims that the defendant is liable for failing to control the intentional conduct of others, in which courts have been more stringent about inferences of causation. See, e.g., Saelzler v. Advanced Group 400, 23 P.3d 1143 (Cal.2001) (plaintiff, who was sexually assaulted by unknown assailants, alleged defendants failed to provide adequate security at apartment building known to be the scene of recurring criminal activity; although defendant's negligence in failing to provide adequate security increased the general risk of such attacks, plaintiff's failure to provide better evidence of the way in which improved security would have prevented assault justified summary judgment on factual cause) (citing similar cases); Mitchell v. Pearson Enters., 697 P.2d 240 (Utah 1985).

Only a handful of courts have addressed the question of whether a presumption of causation exists in negligence per se cases. That may be because the more confined safety concerns reflected in a statute rarely pose difficult causation questions when an actor violates the statute and causes harm. Justice Cardozo first raised the possibility of a presumption of causation based on negligence per se in the classic case of Martin v. Herzog, 126 N.E. 814 (N.Y.1920), in which the plaintiff failed to comply with a statute requiring lights on vehicles after sundown. Declaring that failure to comply with such a statutory obligation was negligence per se, Cardozo proceeded to the question of causation: "We think, however, that evidence of a collision occurring more than an hour after sundown between a car and an unseen buggy, proceeding without lights, is evidence from which a causal connection may be inferred between the collision and the lack of signals." Id. at 816. Cardozo plainly invokes a permissive inference rather than a presumption, although later in the opinion he states, inconsistently, that the burden of demonstrating that lights would not have mattered falls on the defendant. His language leaves substantial ambiguity about

whether the decision is specific to the facts of a vehicle without lights at night or is more generally applicable to violations of safety statutes.

For courts that apply a presumption of causation when defendant's negligence consists of violating a safety statute, see Transorient Navigators Co. v. M/S Southwind, 714 F.2d 1358, 1368 (5th Cir.1983); Bowman v. Redding & Co., 449 F.2d 956, 964-965 (D.C. Cir.1971); Elliott v. Michael James Inc., 559 F.2d 759, 763 (D.C.Cir.1977); Limehouse v. S. Ry. Co., 58 S.E.2d 685, 686 (S.C.1950); see also The Pennsylvania, 86 U.S. 125, 135 (1874) (violation of statutory safety rules in admiralty imposes strong burden on defendant to show that such violation was not the cause of harm); cf. Zuchowicz v. United States, 140 F.3d 381, 390-391 (2d Cir.1998) (Federal Tort Claims Act case adopting Connecticut law). But see Graham v. Am. Cyanamid Co., 350 F.3d 496 (6th Cir.2003) (plaintiff claiming violation of FDA regulations constituted negligence per se nevertheless had the burden of proving causation); Twyman v. Johnson, 655 A.2d 850, 852 (D.C.Ct.App.1995); Merchants Mut. Ins. Co. v. Baker, 473 N.E.2d 827, 828 (Ohio 1984); Yap v. ANR Freight Sys., Inc., 789 S.W.2d 424 (Tex.Ct.App.1990); Schlimmer v. Poverty Hunt Club, 597 S.E.2d 43 (Va.2004); cf. Fagerquist v. W. Sun Aviation, Inc., 236 Cal. Rptr. 633, 643 (Ct.App.1987) (holding that a jury instruction that shifted the burden from the plaintiff to the defendant if the jury found the defendant violated the safety regulations was improper); Bell v. Carson, 270 P.2d 420, 422 (Idaho 1954) (stating that violating a traffic law will not constitute contributory negligence that would bar recovery unless the violation constitutes a proximate cause of the injury); Oxedine v. Lowry, 133 S.E.2d 687, 691 (N.C.1963) (stating that negligence per se can raise no presumption of proximate cause). Canadian law requires the plaintiff to prove causation even in a negligence per se case. See Glanville Williams, *The Effect of Penal Legislation in the Law of Torts*, 23 MOD. L. REV. 233, 234-235 (1960).

The "*Pennsylvania* rule" provides that in a seaman's suit against the employer, a presumption of causation applies, requiring the employer to prove not only that its negligence was most probably not the cause, but that "it could not have been." This rule derives from a 19th-century admiralty decision by the Supreme Court in The Pennsylvania, 86 U.S. (19 Wall.) 125 (1873). Its treatment has been checkered, with courts describing it as an unusual rule peculiar to admiralty law, see Dir. Gen. of India Supply Mission v. S.S. Maru, 459 F.2d 1370, 1375 (2d Cir.1972); Pierro v. Carnegie-Illinois Steel Corp., 186 F.2d 75, 78 (3d Cir.1950), employing it in cases in which there is circumstantial evidence that would support an inference of causation, see Wilkins v. Am Exp. Isbrandtsen Lines, Inc., 446 F.2d 480, 485 (2d Cir.1971), and limiting it on a variety of rationale, see Jones v. Spentonbush-Red Star Co., 155 F.3d 587, 594-596 (2d Cir.1998) (OSHA violations insufficient to invoke the *Pennsylvania* rule based on lack of Congressional intent in enacting OSHA for such a burden shift); and refusing to employ it in cases in which there is substantial doubt about whether a causal relationship exists, see Wills v. Amerada Hess Corp., 379 F.3d 32 (2d Cir.2004) (seaman who died of cancer sued employer for exposure to petrochemicals alleged to have caused cancer).

For courts that apply a presumption of causation in warnings cases, see Benjamin J. Jones, Annotation, *Presumption or Inference, in Products Liability Action Based on Failure to Warn, That User of Product Would Have Heeded an Adequate Warning Had One Been Given*, 38 A.L.R.5th 683 (1996) (listing 17 jurisdictions that have applied a presumption of causation in inadequate-warnings cases); see also Mark Geistfeld, *Inadequate Product Warnings and Causation*, 30 U. MICH J.L. REFORM 309 (1997) (advocating heeding presumption in inadequate-warnings cases).

A prominent case in which a court shifted the burden of proof on causation to defendants is Anderson v. Somberg, 338 A.2d 1 (N.J.1975); see also Chin v. St. Barnabas Med. Ctr., 734 A.2d 778 (N.J.1999). In *Anderson*, a forceps-like medical device broke during an operation and a piece of metal lodged in the patient's spinal column, requiring subsequent surgery and causing substantial injury. The court isolated the cause of the accident to malpractice by the surgeon, a defective medical device sold by the manufacturer or supplier, or inadequate maintenance or inspection of the device in the hospital. *Anderson* recalls the classic case of Ybarra v. Spangard, 154 P.2d 687 (Cal.1944), in which the court applied res ipsa loquitur to several health-care providers and the hospital for an iatrogenic injury suffered by the plaintiff during surgery. The effect of *Ybarra* was to impose the burden of proof on defendants to exonerate themselves or identify the defendant whose negligence caused the plaintiff's harm. *Ybarra* has not been well received, either for application to its specific medical context or for broader application. See Raber v. Tumin, 226 P.2d 574, 579-580 (Cal.1951) (Traynor, J., dissenting) (cautioning on the evils of extending *Ybarra* outside the medical-malpractice context); Spannaus v. Otolaryngology Clinic, 242 N.W.2d 594 (Minn.1976) (refusing to adopt *Ybarra*); Barrett v. Emanuel Hosp., 669 P.2d 835 (Or.Ct.App.1983) (refusing to adopt *Ybarra*); see generally DAN B. DOBBS, THE LAW OF TORTS § 249, at 652 (2000). These cases are rare. See Anderson v. Picciotti, 676 A.2d 127, 135 (1996) ("In a few exceptional cases ... the burden of proof on some issues may shift to the defendant.").

Professor David Robertson provides a cogent explanation for the reason courts have kept the bar for proving causation at only a modest height in David W. Robertson, *The Common Sense of Cause in Fact*, 75 TEX. L. REV. 1765, 1774- 1775 (1997) ("The central idea is that when a defendant has engaged in conduct that we consider to be wrongful in major part because such conduct often leads to the kind of harm the plaintiff has suffered, we are rightfully impatient with the defendant's claim that plaintiff cannot prove that the conduct caused the harm on this occasion."); see also Kwasny v. United States, 823 F.2d 194 (7th Cir.1987) ("The general tendency of courts in tort cases, once negligence is established, is to resolve doubts about causation, within reason, in the plaintiff's favor."); Charles E. Carpenter, *Concurrent Causation*, 83 U. PA. L. REV. 941, 943 (1935) ("The policy of not permitting a wrongdoer to escape liability for a consequence which his wrongful conduct contributed to produce might very well induce the courts in such cases to relax the requirement of proof of causation by a preponderance of the evidence."). Other, more specific policies may affect the threshold of evidence of causation required. For example, courts have understood the Federal Employers Liability Act, 45 U.S.C. § 51, which covers injured employees of interstate railroads, as being solicitous of employees and easing their burden of proof. See, e.g., Gallick v. Balt. & Ohio R.R., 372 U.S. 108, 116-117 (1963) ("It does not matter that, from the evidence, the jury may also with reason, on grounds of probability, attribute the result to other causes.... Judicial appraisal of the proofs to determine whether a jury question is presented is narrowly limited to the single inquiry whether, with reason, the conclusion may be drawn that negligence of the employer played any part at all in the injury or death." (quoting Rogers v. Mo. Pac. R.R. Co., 352 U.S. 500, 506-507 (1957)); Aparicio v. Norfolk & W. Ry. Co., 84 F.3d 803, 810 (6th Cir.1996) (plaintiff in an FELA case must "present more than a scintilla of evidence in order to create a jury question on the issue of employer liability, but not much more"). *Rogers* and *Gallick* and their treatment of factual cause in FELA cases can only be understood as easing a plaintiff's burden of proving causation, not modifying the requirement of factual causation. As explained in § 26, Comment *j*, causation is a

concept that exists or not; it is not a matter of degree.

The policy ground for liberality in judging plaintiff's evidence of causation is subject to at least two caveats. First, in some cases, the evidence of negligence will not be independent of the evidence of causation. Thus, when plaintiff alleges that a pharmaceutical manufacturer failed to warn of the risk of a serious adverse side effect and the capacity of the drug to cause that side effect is in dispute, there is little call for leniency with regard to plaintiff's evidence of the capacity of the drug to cause the side effect. Second, when the plaintiff's own contributory fault is also a cause of the harm, the equities of the situation do not so strongly favor the plaintiff; many of the burden-shifting or-modifying rules with regard to proof of causation were crafted when contributory negligence was a complete bar to recovery, and culpable plaintiffs were not able to recover from others.

For recognition that merely increasing the risk of harm is not sufficient in all cases to satisfy the burden of proof, see Fedorczyk v. Caribbean Cruise Lines, Ltd., 82 F.3d 69, 76 (3d Cir.1996) (applying New Jersey law) ("Simply put, increased risk of harm due to defendant's negligence, standing alone, does not permit an inference that an injury, more probably not, was caused by the negligence."); cf. Mario J. Rizzo, *Foreword: Fundamentals of Causation,* 63 CHI.-KENT L. REV. 397, 403 (1987) ("A rise in the probability (frequency) of an outcome may be evidence of causation. It is not the causal phenomenon itself.") (footnote omitted). Slightly more precisely, not every case of increased risk permits such an inference; nevertheless, circumstantial evidence varies and provides inferences of varying strength that must be evaluated based on the specific facts of the case and policies entailed in the substantive claim. See Liriano v. Hobart Corp., 170 F.3d 264, 271 (2d Cir.1999) (applying New York law) ("When a defendant's negligent act is deemed wrongful precisely because it has a *strong* propensity to cause the type of injury that ensued, that very causal tendency is evidence enough to establish a prima facie case of cause-in-fact.") (emphasis added).

Comment c. Toxic substances and disease. This Comment and these Reporters' Notes benefited significantly from a review of a prior draft by a panel consisting of prominent epidemiologists and a physician that was assembled by the Science, Technology, and Law Program of the National Academy of Sciences. The panelists included Dr. Steven Goodman, of Johns Hopkins University, Dr. Leon Gordis, also of Johns Hopkins University, Dr. Jerome Kassirer, of Tufts and Yale Universities, Dr. David Savitz, of the University of North Carolina, and Dr. Douglas Weed, of the National Cancer Institute, and were selected by the Science, Technology, and Law Program based on relevant expertise, familiarity with the use of scientific evidence in law, and their independence and objectivity. A meeting took place on January 21, 2003, at the National Academy of Sciences and included the Reporters for this Restatement, several others from the ALI, and the panelists. The meeting consisted of a very productive dialogue among the panelists and Institute representatives about the prior draft of this Comment and Reporters' Notes. A transcript of the meeting is available on the website of the Science, Technology, and Law Program of the National Academies of Science at: http://www7.nationalacademies.org/stl/index.html. The Institute and the Reporters are indebted to the panelists for their valuable contributions, as well as to Professor Richard Merrill, Co-Chair of the Science, Technology, and Law Panel and Professor of Law at the University of Virginia, who understood the benefit such a meeting might have and graciously endeavored both to make it possible and oversee the necessary arrangements, and Dr. Anne-Marie Mazza, of the National Academy of Sciences staff, who cheerfully and energetically attended to the arrangements for the

meeting. The Institute and Reporters also thank the Science, Technology, and Law Program for its cooperation in making this meeting possible and ALI member Patrick Malone who first suggested such a joint effort.

(1) *Introduction.* Since the mid-1970s when asbestos litigation began, there has been a steady stream of toxic-substances litigation. Some of it is large-scale, exemplified by such well-known case congregations as asbestos, Agent Orange, DES, Bendectin, silicone-gel breast implants, and fen-Phen. There are also more limited or localized cases, such as hazardous-waste cases. In addition to agents such as those identified above, an activity, such as continual use of a keyboard, may be responsible for a person's disease. These cases require courts to adapt traditional rules of proof to the greater uncertainty inherent in agent-disease causation and the specialized types of evidence that may be available. The relative absence of knowledge about the mechanisms involved in disease causation, compared to more traditional traumatic injuries, gets to the core of the adjustments required: "Scientists know very little about how, in a mechanistic sense, toxic substances cause disease such as cancer or birth defects. Nonetheless, they may know a considerable amount about whether toxic substances cause disease or injury through inferences drawn from statistical associations and other indirect means." Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH. L.J. 189, 209-210 (1992) (emphasis and footnotes omitted). For thumbnail sketches of most of the major toxic-substances litigations of the 20th century and the scientific evidence of causation available in each, see Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience*, 18 COLUM. J. ENVTL. L. 181, 279-326 (1993). For an interesting illustration of this distinction, in which a court required expert testimony to establish the causal connection between an accident and a chronic condition but did not require such testimony for the immediate, traumatic consequence of the accident, see Dodge-Farrar v. Am. Cleaning Servs. Co., 54 P.3d 954 (Idaho Ct.App.2002).

Toxic-substances litigation was also the genesis for substantial reform in the law governing the admissibility of expert-witness testimony. Bendectin provided the occasion for the Supreme Court's initial foray into the field in Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993), and a hazardous-waste case occasioned the next Supreme Court decision in the area, General Electric Co. v. Joiner, 522 U.S. 136 (1997). A considerable body of federal law now exists in which *Daubert* and its progeny have been applied to expert witnesses who propose to testify on some aspect of agent-disease causation. Many state courts have followed the approach of the federal courts in addressing these matters through the admissibility lens. See, e.g, Logerquist v. McVey, 1 P.3d 113 (Ariz.2000); Goeb v. Thraldson, 615 N.W.2d 800 (Minn.2000); In re Canavan, 733 N.E.2d 1042 (Mass.2000); Schafersman v. Agland Coop., 631 N.W.2d 862 (Neb.2001). The federal courts have not achieved perfect consistency in all aspects of their admissibility law and decisions. See, e.g., Jerome P. Kassirer & Joseph S. Cecil, *Inconsistency in Evidentiary Standards for Medical Testimony: Disorder in the Courts*, 288 JAMA 1382 (2002). State courts reveal even greater variation in this area. However, whether they formally adopt *Daubert* as the standard governing expert-witness testimony, continue to adhere to the standard provided in Frye v. United States, 293 F. 1013 (D.C.Cir.1923), or decide to employ some modification of these standards, state courts have been increasingly confronted with the question of adequate proof of toxic causation and the admissibility of expert-witness testimony on that subject. And regardless of the rules adopted for admitting expert testimony, the principles set forth in Comment *c* are frequently reflected in state-court decisions. See Lofgren v.

Motorola, 1998 WL 299925 (Ariz.Super.Ct. June 1, 1998); U.S. Sugar Corp. v. Henson, 823 So. 2d 104 (Fla.2002); Berry v. CSX Transp., Inc., 709 So. 2d 552 (Fla.Dist.Ct.App.1998); Donaldson v. Cent. Ill. Pub. Serv. Co., 767 N.E.2d 314 (Ill.2002); Linnen v. A.H. Robins Co., 2000 WL 16769 (Mass.Super.Ct. Dec. 14, 1999); McDonough v. Allina Health Sys., 685 N.W.2d 688 (Minn.Ct.App.2004); Schafersman v. Agland Coop., 631 N.W.2d 862 (Neb.2001); Valentine v. PPG Indus., Inc., 2004 WL 1908303 (Ohio Ct.App.2004); Cutlip v. Norfolk S. Corp., 2003 WL 1861015 (Ohio Ct.App.2003); Jennings v. Baxter Healthcare Corp., 14 P.3d 596 (Or.2000); McDaniel v. CSX Transp., Inc., 955 S.W.2d 257 (Tenn.1997); Merrell Dow Pharms., Inc. v. Havner, 953 S.W.2d 706, 718 (Tex.1997); Easum v. Miller, 92 P.3d 794 (Wyo.2004); see also Kuhn v. Sandoz Pharms. Corp., 14 P.3d 1170, 1184-1185 (Kan.2000) (declining to require proof of general causation in a case in which there was not mass exposure and an absence of a body of epidemiologic evidence). Indeed, the federal courts, which were the first to confront these issues, are frequently cited by their state counterparts. See, e.g., John's Heating Serv. v. Lamb, 46 P.3d 1024, 1033-1037 (Alaska 2002); Kaelbel Wholesale, Inc. v. Soderstrom, 785 So. 2d 539 (Fla.Dist.Ct.App.2001); DePyper v. Navarro, 1995 WL 788828 (Mich.Cir.Ct. Nov. 27, 1995); Allison v. Fire Ins. Exch., 98 S.W.3d 227 (Tex.Ct.App.2002); Neal v. Dow Agrosciences LLC, 74 S.W.3d 468 (Tex.Ct.App.2002); Easum v. Miller, 92 P.3d 794 (Wyo.2004) (addressing the reliability of differential diagnoses to determine specific causation). For a cataloguing of the general approach to admissibility of expert testimony in the states, see *States Move to* Daubert, *Even When They Say They're Stuck on* Frye, 30 BNA PROD. SAFETY & LIAB. REP. 328 (2002); Leo H. Whinery, *Expert Testimony Trends in State Practice and the Uniform Rules of Evidence*, in New Directions in Expert Testimony: Scientific, Technical, and Other Specialized Knowledge Evidence in Federal and State Courts 151, 176-182 (ALI-ABA Course of Study, Apr. 26, 2001); Edward J. Imwinkelried, *Evidentiary Balance*, Nat'l L.J., May 13, 2002, at B11 ("commenting that [i]t would be a mistake to overstate the differences between the jurisdictions following *Frye* and those committed to *Daubert*," and observing that the commonest explanation for a state court declining to adopt *Daubert* is because it is too liberal in permitting expert testimony to be admitted). This Comment does not address nor attempt to resolve the appropriate standard for determining the admissibility of expert testimony on agent-disease causation. Expert-witness testimony is employed to prove agent-disease causation, and the admissibility of an expert's opinion may be determinative as to whether the plaintiff satisfies the burden of production on agent-disease causation. Yet the emphasis on greater scrutiny of expert-witness testimony has been with regard to the basis of the expert's opinion (in addition to the methodology and reasoning employed by the expert) thereby resulting in an examination of the scientific evidence that exists to support the expert's opinion. Courts frequently assess the state of the scientific record and only when it meets a sufficiency threshold is an expert witness permitted to testify about the existence of agent-disease causation. See, e.g., Wills v. Amerada Hess Corp., 379 F.3d 31 (2d Cir.2004); Vargas v. Lee, 317 F.3d 498 (5th Cir.2003) (concluding absence of scientific studies connecting trauma to fibromyalgia required ruling expert's contrary testimony inadmissible); In Re: Silicone Gel Breasts Implants Products Liability Litig., 318 F. Supp. 2d 879, 898-899 (C.D.Cal.2004) (excluding expert's testimony about a "suggestive" connection between defendant's agent and cancer because study on which expert relied did not support such a conclusion); Newman v. Motorola, Inc., 218 F. Supp. 2d 769 (D.Md.2002) (absence of reliable evidence of connection between cellular phones and cancer fatal to admissibility of expert's testimony); Lindquist v. City of Jersey City Fire Dep't, 2002 NJ LEXIS 25 (N.J.2003) (examining

the scientific evidence upon which expert's opinion was based); Brookshire Brothers, Inc. v. Smith, 176 S.W.3d 30, 2004 WL 1064776 (Tex.App.2004); Daniels v. Lyondell-Citgo Refining Co., 99 S.W.3d 722 (Tex.App.2003) (reviewing 3 epidemiologic studies on which plaintiffs' experts relied for their opinion and concluding that none was sufficient to support a finding of causation); see also Exxon Corp. v. Makofski, 116 S.W.3d 176 (Tex.Ct.App.2003) (examining scientific studies and evidence relied on by experts in determining the sufficiency of the evidence on causation).

Among the most useful sources explaining the various scientific fields applicable to proving causation and illustrating their application to toxic-substances litigation are FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed. 2000); DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY (2d ed. 2002). For a useful summary and synthesis of the Supreme Court's decisions on the admissibility of expert testimony, see Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107, 112- 119 (2001).

For explanations of some of the differences in characteristics of toxic agents that bear on the differences in proof of causation, see Daniel Farber, *Toxic Causation*, 71 MINN. L. REV. 1219, 1251-1259 (1987); Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience*, 18 COLUM. J. ENVTL. L. 181 (1993); Michael D. Green, *The Paradox of Statutes of Limitations in Toxic Substances Litigation*, 76 CALIF. L. REV. 965, 972-976 (1988).

For a statement of the trilogy of elements in proof of agent-disease causation--namely, exposure, general causation, and specific causation--see Stevens v. Sec'y of HHS, 2001 WL 387418, at *35 (Fed.Cl. March 30, 2001). *Stevens* also contains an excellent explanation of the difficulties and extensive efforts required to resolve questions of agent-disease causation in the National Vaccine Injury Compensation Program. Id. at *7.

For an example of the sort of critical assessment scientists might have for specific scientific testimony in court mentioned in Comment *c(1)*, see Sander Greenland, *The Need for Critical Appraisal of Expert Witnesses in Epidemiology and Statistics*, 39 WAKE FOREST L. REV. 291 (2004).

(2) *Exposure to the agent.* In connection with proof of either specific or general causation, the extent of the victim's exposure to an agent, i.e., the dose, may play a significant role in determining causation. Dose includes both the intensity of exposure and the duration, although for different agents and diseases, one or the other may be more significant. Dosage may be important for two reasons: 1) for most agent-disease relationships, the higher the dose, the greater the risk of disease; 2) for some diseases there may be a threshold dose; exposures to the agent below the threshold dose pose no identifiable risk of causing the disease. See MICHAEL A. KAMRIN, TOXICOLOGY: A PRIMER ON TOXICOLOGY PRINCIPLES AND APPLICATIONS 36-38 (1988); Kenneth J. Rothman, *Causes*, 104 Am. J. Epidemiology 587 (1976) (accounting for dose-response relationships with an explanatory causal model). On dose-response relationships, see DAVID E. LILIENFELD & PAUL STOLLEY, FOUNDATIONS OF EPIDEMIOLOGY 265 (3d ed. 1994); RICHARD K. RIEGELMAN & ROBERT P. HIRSCH, STUDYING A STUDY AND TESTING A TEST: HOW TO READ THE HEALTH SCIENCE LITERATURE 45 (3d ed. 1996); Gerald W. Boston, *A Mass-Exposure Model of Toxic*

Causation: The Content of Scientific Proof and the Regulatory Experience, 18 COLUM. J. ENVTL. L. 181, 215-217 (1993). The existence of a threshold dose before an effect can occur is a controversial concept for which current scientific thinking resists any universal answers and instead examines what is known about the pathological mechanisms of the disease. See Proposed Guidelines for Carcinogen Risk Assessment, 61 Fed. Reg. 17,960, 17,993 (E.P.A.1996). Compare Marvin Goldman, *Cancer Risk of Low-Level Exposure*, 271 SCI. 1821 (1996) (challenging the conventional wisdom that cancer risk is not subject to a threshold dose); Bruce N. Ames, Renae Magaw & Lois Swirsky Gold, *Ranking Possible Carcinogenic Hazards*, 236 SCI. 271 (1987) (arguing that animal toxicology studies that support no threshold cannot be extrapolated to humans) with R.W. Hart & L. Fishbein, *Interspecies Extrapolation of Drug and Genetic Toxicity Data*, in 1 TOXICOLOGICAL RISK ASSESSMENT 3, 32 tbl. 19 (D.B. Clayson et al. eds., 1985) (arguing against threshold-dose theory).

For cases in which unknown dose or insufficient proof of dose played a critical role, see Mitchell v. Gencorp Inc., 175 F.3d 778, 781 (10th Cir.1999); Christopherson v. Allied-Signal Corp., 939 F.2d 1106 (5th Cir.1991) (occupational exposure to nickel/cadmium batteries) (applying Texas law); Martin v. Shell Oil Co., 180 F. Supp. 2d 313, 318-319 (D.Conn.2002) (expert's explanation of how chemical migrated to plaintiff's property did not require testing where cost of test was in the range of $100,000); In re Three Mile Island Litig. Consol. Proceedings, 927 F. Supp. 834, 870 (M.D.Pa.1996), aff'd in relevant part, 193 F.3d 613 (3d Cir.1999); Cavallo v. Star Enter., 892 F. Supp. 756 (E.D.Va.1995), aff'd in part and rev'd in part, 100 F.3d 1150 (4th Cir.1996); Whiting v. Boston Edison Co., 891 F. Supp. 12 (D.Mass.1995); Schmaltz v. Norfolk & W. Ry. Co., 878 F. Supp. 1119 (N.D.Ill.1995); Wade-Greaux v. Whitehall Labs., 874 F. Supp. 1441 (D.V.I.1994); Renaud v. Martin Marietta Corp., 749 F. Supp. 1545, 1555 (D.Colo.1990) (exposure to contaminated water supply alleged to cause cancer and other diseases), aff'd, 972 F.2d 304 (10th Cir.1992); John's Heating Serv. v. Lamb, 46 P.3d 1024 (Alaska 2002); Alder v. Bayer Corp., 61 P.3d 1068, 1085-1088 (Utah 2002) (summarizing other opinions on the precision with which plaintiffs must prove the dosage to which they were exposed); see also Eagle-Picher v. Balbos, 578 A.2d 228, 245 (Md.Ct.App.1990) (recognizing that dose of asbestos exposure required to cause mesothelioma is considerably lower than to cause asbestosis or lung cancer).

In cases involving occupational exposure to asbestos, many courts have fashioned a "frequency, regularity, and proximity" standard, requiring a plaintiff to prove these elements for each defendant's asbestos products in order adequately to establish exposure to a defendant's asbestos product. See Jackson v. Anchor Packing Co., 994 F.2d 1295 (8th Cir.1993) (applying Arkansas law); Slaughter v. S. Talc Co., 949 F.2d 167 (5th Cir.1991) (applying Texas law); Hoffman v. Allied Corp., 912 F.2d 1379 (11th Cir.1990) (applying Florida law); Menne v. Celotex Corp., 861 F.2d 1453 (10th Cir.1988) (applying Nebraska law); Thompson v. S. Pac. Transp. Co., 809 F.2d 1167 (5th Cir.1987) (applying Louisiana law); Lohrmann v. Pittsburgh Corning Corp., 782 F.2d 1156, 1162-1163 (4th Cir.1986) (applying Maryland law); Blackston v. Shook & Fletcher Insulation Co., 764 F.2d 1480 (11th Cir.1985) (applying Georgia law); Donaldson v. Cent. Ill. Pub. Serv. Co., 767 N.E.2d 314 (Ill.2002); Sholtis v. Am. Cyanamid Co., 568 A.2d 1196 (N.J.Super.Ct.App.Div.1989); Eckenrod v. GAF Corp., 544 A.2d 50 (Pa.Super.Ct.1988); see also James v. Bessemer Processing Co., Inc., 714 A.2d 898 (N.J.1998) (applying "frequency, regularity, and proximity" test to chemicals alleged to cause cancer); Jobe v. W.P. Metz Refining, 664 A.2d 1015, 1020 (Pa.Super.Ct.1995) (applying same test to occupational exposure to

cadmium, alleged to have caused plaintiff's cancer).
For courts confronting the problem of exposure in a hazardous-waste case, see In re Paoli R.R. Yard PCB Litig., 916 F.2d 829 (3d Cir.1990); In re TMI Litig., 193 F.3d 613 (3d Cir.1997) (lengthy opinion addressing admissibility of expert witnesses testifying about dose/exposure arising out of nuclear-power-plant accident); see also Sandra A. Geschwind et al., *Risk of Congenital Malformations Associated with Proximity to Hazardous Waste Sites*, 135 AM. J. EPIDEMIOLOGY 1197 (1992) (explaining difficulties of studying effects of hazardous-waste sites); Leon Gordis, *Epidemiologic Approaches for Studying Human Diseases in Relation to Hazardous Waste Disposal Sites*, 25 HOUS. L. REV. 837 (1988); Paul J. Lioy, *The Analysis of Total Human Exposure for Exposure Assessment: A Multi-Discipline Science for Examining Human Contact with Contaminants,* 24 ENVTL. SCI. & TECH. 938 (1990). Frequently, proof of exposure in these cases is based on modeling designed to estimate exposure. As with other types of scientific studies that may be used to demonstrate general causation, these models may be based on carefully collected and robust data and accepted or well-conceived assumptions that have some validation and therefore are of considerable evidentiary value, or they may be based on questionable assumptions not subjected to any efforts at validation and therefore of little probative value. See Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH. L.J. 189, 237-240 (1992).
Infrequently, toxic agents produce a specific biomarker in those who have been exposed to the agent. Identification of the biomarker in an individual then indicates exposure to the agent and may, in some cases, permit an assessment of the dosage to which an individual was exposed. Gary E. Marchant, *Genetic Susceptibility and Biomarkers in Toxic Injury Litigation*, 41 JURIMETRICS J. 67, 68, 73-74, 95-97 (2000) (explaining concept of biomarkers, how they might be used to provide evidence of exposure or dose, discussing cases in which biomarkers were invoked in an effort to prove exposure, and concluding, "biomarkers are likely to be increasingly relied on to demonstrate exposure").
(3) *General causation.* The concepts of general causation and specific causation are widely accepted among courts confronting causation issues with toxic agents. See, e.g., Kelley v. Am. Heyer-Schulte Corp., 957 F. Supp. 873, 875- 876 (W.D.Tex.1997) (recognizing the different concepts of general and specific causation), appeal dismissed, 139 F.3d 899 (5th Cir.1998); Cavallo v. Star Enter., 892 F. Supp. 756, 771 n.34 (E.D.Va.1995), aff'd in part and rev'd in part, 100 F.3d 1150 (4th Cir.1996); Casey v. Ohio Med. Prods., 877 F. Supp. 1380, 1382 (N.D.Cal.1995); Merrell Dow Pharms., Inc. v. Havner, 953 S.W.2d 706, 714-715 (Tex.1996). But see Donaldson v. Cent. Ill. Pub. Serv. Co., 767 N.E.2d 314 (Ill.2002) (rejecting use of "generic" and specific causation; plaintiff need only prove cause in fact).
When the connection between an agent and disease is strong and well documented, general-causation issues fade into the background. Thus, in asbestos cases, the general-causation question does not arise with regard to mesothelioma, asbestosis, and lung cancer because the causal connection between asbestos and those diseases is quite well established. See, e.g., Karjala v. Johns-Manville Prod. Corp., 523 F.2d 155, 160 (8th Cir.1975) (applying Minnesota law); Bertrand v. Johns-Manville Sales Corp., 529 F. Supp. 539, 544 (D.Minn.1982) ("[I]t is clear that it is appropriate to estop litigation on the issue of whether asbestos dust can cause diseases such as asbestosis and mesothelioma. This proposition is so firmly entrenched in the medical and legal literature that it is not subject to serious dispute."); Flatt v. Johns-Manville Sales Corp., 488 F. Supp. 836, 841 (E.D.Tex.1980) (holding that asbestos exposure causes asbestosis and

mesothelioma, as a matter of law). Although general causation may not be an issue for one or several diseases caused by an agent such as asbestos, general causation may be an issue with regard to other diseases, as is the case with asbestos and colon and gastrointestinal cancers. See In re Joint E. & S. Dist. Asbestos Litig., 52 F.3d 1124 (2d Cir.1995) (applying New York law); Landrigan v. Celotex Corp., 605 A.2d 1079 (N.J.1992); Grassis v. Johns-Manville, 591 A.2d 671 (N.J.Super.Ct.App.Div.1991).

Occasionally, an ailment may be so strongly associated with a specific agent and so rarely (if ever) associated with any other cause that it is called a "signature disease." Examples of signature diseases are vaginal adenocarcinoma in the daughters of mothers exposed to DES and asbestosis in those exposed to asbestos. Once a signature disease is identified, there is no need for proof of either general causation or specific causation, as the existence of the disease is tied to exposure to the signature agent. See Daniel A. Farber, *Toxic Causation*, 71 MINN. L. REV. 1219, 1251-1252 (1987); Kenneth S. Abraham & Richard A. Merrill, *Scientific Uncertainty in the Courts*, ISSUES SCI. & TECH., Winter 1986, at 93, 101.

Cases involving signature diseases are, however, rare. In cases in which group studies are employed as proof, proof of causation proceeds in two steps: general causation and specific causation. Cases accepting the proposition that relevant epidemiologic studies are acceptable evidence to support proof of general causation are legion. See, e.g., Smith v. Ortho Pharm. Corp., 770 F. Supp. 1561, 1571 (N.D.Ga.1991) (explaining increased reliance of courts on epidemiologic evidence in toxic-substances litigation); Stevens v. Sec'y of HHS, 2001 WL 387418, at *12-13 (Fed.Cl. March 30, 2001); James v. Chevron U.S.A., Inc., 694 A.2d 270, 280 (N.J.Super.Ct.App.Div.1997), aff'd, 714 A.2d 898 (N.J.1998); see generally Michael D. Green et al., *Reference Guide on Epidemiology*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 335 n.2 (2d ed. 2000).

However, even when epidemiology finds an association, the observational (rather than experimental) nature of these studies requires an examination of whether the association is truly causal or spurious and due to random error or deficiencies in the study (bias). The same problems may produce a study that does not find an association when there truly is a causal relationship between the agent and the disease in question. See Michael D. Green et al., *Reference Guide on Epidemiology*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 374-375 (2d ed. 2000); Berry v. CSX Transp., Inc., 709 So. 2d 552, 558 (Fla.Dist.Ct.App.1998); Schafersman v. Agland Coop., 631 N.W.2d 862, 871 (Neb.2001).

Criteria for assessing whether an association is causal were proposed by Sir Austin Bradford Hill. One formulation of these criteria is:

(1) Is the temporal relationship correct? Does the "effect" follow the "cause"?
(2) Is there evidence from true experiments in humans?
(3) Is the association a strong one?
(4) Is the association consistent from study to study?
(5) Is there a dose-response gradient?
(6) Is the association specific?
(7) Does the association make biological sense?
(8) Is there an appropriate analogy to other known causal relationships?

See Austin Bradford Hill, *The Environment and Disease: Association or Causation*?, 58 PROC. ROY. SOC. MED. 295 (1965). For discussion of these criteria and their respective strengths in informing a causal inference, see 2 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC

EVIDENCE § 28-2.2.3 (1997); LEON GORDIS, EPIDEMIOLOGY 176-181 (1996); DAVID E. LILIENFELD & PAUL D. STOLLEY, FOUNDATIONS OF EPIDEMIOLOGY 263-266 (3D ED. 1994); DOUGLAS L. WEED, EPIDEMIOLOGIC EVIDENCE AND CAUSAL INFERENCE, 14 Hematology/Oncology Clinics N. Am. 797 (2000). FOR A DEFINITION OF AND CRITICAL INQUIRY INTO WHAT IS MEANT BY THE SEVENTH CRITERION, BIOLOGIC PLAUSIBILITY, SEE DOUGLAS L. WEED & STEPHEN D. HURSTING, BIOLOGIC PLAUSIBILITY IN CAUSAL INFERENCE: CURRENT METHODS AND PRACTICE, 147 Am. J. Epidem. 415 (1998) (EXAMINING USE OF THIS CRITERION IN CONTEMPORARY EPIDEMIOLOGIC RESEARCH AND DISTINGUISHING BETWEEN A PLAUSIBLE HYPOTHESIS AND ONE SUPPORTED BY EVIDENCE SUPPLIED FROM RESEARCH EMPLOYING MOLECULAR BIOLOGY AND MOLECULAR EPIDEMIOLOGY).

The first case to employ an epidemiologic threshold for proof of agent-disease causation was Brock v. Merrell Dow Pharms., 874 F.2d 307, 315 (5th Cir.) (applying Texas law), modified on rehearing, 884 F.2d 166 (5th Cir.1989). The genesis for that requirement was the Bendectin litigation in which, in the face of a developing body of scientific evidence tending to exonerate Bendectin, courts sought a means to prevent submission of those cases to a jury. When a substantial body of epidemiologic evidence exists that tends to exonerate the alleged agent, other evidence of causation is far less persuasive. The Bendectin and silicone-gel breast-implant cases, the latter of which involve autoimmune diseases, teach this lesson. Earlier, in In re "Agent Orange" Products Liability Litigation, 611 F. Supp. 1267 (E.D.N.Y.1985), Judge Weinstein had denigrated the animal studies on which plaintiffs sought to rely in the course of granting defendants summary judgment, thereby implying that epidemiologic evidence would be required. After *Brock*, several district courts in the Fifth Circuit employed it as a precedent, requiring epidemiologic evidence, and courts have used a variety of techniques to squelch Bendectin plaintiffs in the face of a substantial body of exonerative epidemiology. See JOSEPH SANDERS, BENDECTIN ON TRIAL: A STUDY OF MASS TORT LITIGATION 89 (1998) (concluding that "the substantial weight of the scientific evidence fails to support the conclusion that Bendectin causes birth defects"); Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience*, 18 COLUM. J. ENVTL. L. 181 (1993); Michael D. Green, *The Road Less Well Traveled (And Seen): Contemporary Lawmaking in Products Liability*, 49 DEPAUL L. REV. 377 (1999). For applications of the same principle in a non-Bendectin case, see Conde v. Velsicol Chemical Co., 24 F.3d 809 (6th Cir.1994) ("Nineteen epidemiological studies in humans have found little evidence of long-term adverse health effects from chlordane doses hundreds of times higher than those the [plaintiffs] were subject to under a worst-case scenario.").

A quite substantial body of case law and commentary rejects an epidemiologic threshold for sufficient proof of general causation. Many courts find that requiring proof by scientific evidence that does not exist and is not reasonably available to plaintiff when other, reasonably probative evidence exists is an overbroad method for screening cases. See Rider v. Sandoz Pharm. Corp., 295 F.3d 1194, 1198 (11th Cir.2002) ("It is well-settled that while epidemiological studies may be powerful evidence of causation, the lack thereof is not fatal to a plaintiff's case."); Hollander v. Sandoz Pharms. Corp., 289 F.3d 1193, 1211-1212 (10th Cir.2002) (agreeing with the proposition that plaintiffs need not, in all circumstances, provide evidence of general causation with epidemiologic studies); In re Berg Litig., 293 F.3d 1127, 1130 (9th Cir.2002); Bonner v. ISP

Tech., Inc., 259 F.3d 924, 929 (8th Cir.2001); Kennedy v. Collagen Corp., 161 F.3d 1226, 1230 (9th Cir.1998); Zuchowicz v. United States, 140 F.3d 381, 389-390 (2d Cir.1998) (Connecticut law in Federal Tort Claims Act case; acute response to a drug); Ambrosini v. Labarraque, 101 F.3d 129, 138-139 (D.C.Cir.1996) (applying District of Columbia law) (permitting plaintiff's expert to testify in the absence of epidemiological evidence); Benedi v. McNeil-P.P.C., Inc., 66 F.3d 1378 (4th Cir.1995) (applying Virginia law); McCullock v. H.B. Fuller Co., 61 F.3d 1038 (2d Cir.1995) (applying Vermont law); Hopkins v. Dow Corning Corp., 33 F.3d 1116 (9th Cir.1994) (applying California law); Glaser v. Thompson Med. Co., 32 F.3d 969 (6th Cir.1994) (applying Michigan law); Mendes-Silva v. United States, 980 F.2d 1482 (D.C.Cir.1993) (applying District of Columbia law applicable in Federal Tort Claims Act case); Kennedy v. Collagen Corp., 974 F.2d 1342 (9th Cir.1992); Wells v. Ortho Pharm. Corp., 788 F.2d 741, 745 (11th Cir.1986) (applying Georgia law) (pre-*Daubert* case); In re Meridia Prods. Liab. Litig., 328 F. Supp. 2d 791 (N.D. Ohio 2004); Globetti v. Sandoz Pharms., Corp., 111 F. Supp. 2d 1174 (N.D.Ala.2000); Graham v. Playtex Prod., Inc., 993 F. Supp. 127, 132 (N.D.N.Y.1998) (permitting testimony on cause of toxic-shock syndrome in the absence of epidemiological evidence); Lakie v. Smithkline Beecham, 965 F. Supp. 49, 56 (D.D.C.1997) (acknowledging significance of epidemiology but denying its absence is dispositive); Pick v. Am. Med. Sys., Inc., 958 F. Supp. 1151, 1158 (E.D.La.1997) (observing that while epidemiologic studies are a "most useful and conclusive type of evidence," they are not a "necessary element in all toxic tort cases"); Bowers v. N. Telecom, Inc., 905 F. Supp. 1004 (N.D. Fla.1995); Villari v. Terminix Int'l, Inc., 692 F. Supp. 568 (E.D.Pa.1988); Marsee v. U.S. Tobacco Co., 639 F. Supp. 466 (W.D.Okla.1986); Althen v. Sec'y, Dep't of Health & Human Servs., 2003 WL 21439669 (Fed.Cl.2003); Stevens v. Sec'y of HHS, 2001 WL 387418, at *8 (Fed.Cl. March 30, 2001); U.S. Sugar Corp. v. Henson, 823 So. 2d 104 (Fla.2002); Earl v. Cryovac, 772 P.2d 725 (Idaho Ct.App.1989); Donaldson v. Cent. Ill. Pub. Serv. Co., 767 N.E.2d 314 (Ill.2002); Kuhn v. Sandoz Pharms. Corp., 14 P.3d 1170, 1184-1185 (Kan.2000); Bloomquist v. Wapello County, 500 N.W.2d 1, 5 (Iowa 1993); Callahan v. Cardinal Glennon Hosp., 863 S.W.2d 852 (Mo.1993); Lindquist v. City of Jersey City Fire Dep't, 789 A.2d 642 (N.J.2003) (agent-disease causation in workers'- compensation case; supporting the idea that causation should be determined based upon the scientific evidence that is currently available); Rubanick v. Witco Chem. Corp., 593 A.2d 733 (N.J.1991); Valentine v. PPG Indus., Inc., 821 N.E.2d 580, 2004 WL 1908303 (Ohio Ct.App.2004); Jennings v. Baxter Healthcare Corp., 14 P.3d 596 (Or.2001); Reese v. Stroh, 874 P.2d 200 (Wash.Ct.App.1994); Easum v. Miller, 92 P.3d 794 (Wyo.2004); David L. Faigman et al., *How Good Is Good Enough?: Expert Evidence Under* Daubert *and* Kumho, 50 CASE W. RES. L. REV. 645, 663 (2000) ("It is now clear that courts will not exclude causal opinions based on non-epidemiological evidence in situations where a body of such data does not exist."). Commentators have generally been unsympathetic to the imposition of an epidemiologic threshold for proof of causation. See David L. Faigman et al., *How Good Is Good Enough?: Expert Evidence Under* Daubert *and* Kumho, 50 CASE W. RES. L. REV. 645, 663 (2000); Lucinda M. Finley, *Guarding the Gate to the Courthouse: How Trial Judges Are Using Their Evidentiary Screening Role to Remake Tort Causation Rules*, 49 DEPAUL L. REV. 335, 339 (1999); Mark Geistfeld, *Scientific Uncertainty and Causation in Tort Law*, 54 VAND. L. REV. 1011 (2001); Michael D. Green, *Expert Witnesses and Sufficiency of Evidence in Toxic Substances Litigation: The Legacy of* Agent Orange *and Bendectin Litigation*, 86 NW. U. L. REV. 643 (1992); see also Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The*

Content of Scientific Proof and the Regulatory Experience, 18 COLUM. J. ENVTL. L. 181 (1993) (arguing epidemiologic evidence should not be required in cases involving infrequent or isolated exposures but that when large numbers of people are exposed, epidemiologic evidence is and should be required). Even more antithetical to an epidemiologic threshold are commentators who advocate some form of burden shifting on agent-disease causation. See Margaret Berger, *Eliminating General Causation: Notes Towards a New Theory of Justice and Toxic Torts*, 97 COLUM. L. REV. 2117 (1997); Heidi Li Feldman, *Science and Uncertainty in Mass Exposure Litigation*, 74 TEX. L. REV. 1, 45 (1995); Ariel Porat & Alex Stein, *Liability for Uncertainty: Making Evidential Damage Actionable*, 18 CARDOZO L. REV. 1891 (1997); Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773 (1997).

For courts that have confronted the situation in which epidemiologic studies cannot feasibly be conducted because an insufficient number of persons have been exposed, see Dawsey v. Olin, 782 F.2d 1254 (5th Cir.1986) (workers exposed to accidental release of phosgene gas); Donaldson v. Cent. Ill. Pub. Serv. Co., 767 N.E.2d 314 (Ill.2002) (very few cases of disease, neuroblastoma; difficulty in retrospective determinations of exposure); Trach v. Fellin, 817 A.2d 1102 (Pa.Super.Ct.2003) (pharmacy assistant's negligence in providing plaintiff incorrect drug resulted in his taking more than 3 times the maximum recommended dose of drug); Oukrop v. Wasserburger, 755 P.2d 233 (Wyo.1988) (error in prescription resulted in plaintiff being exposed to dose 25 times the ordinary dose). Even when an individual is uniquely exposed to an overdose such as in the *Oukrop* case, studies of the adverse effects of the drug with normal doses may provide evidence supportive of the claim that the overdose caused plaintiff's disease. The studies may not, however, if there is a threshold dose above the therapeutic dose required before the disease can occur in humans or if the incidence of the disease occurring at therapeutic doses is so rare that studies are inadequate to reveal the effect of the agent. Another instance in which epidemiologic studies are inadequate on general causation is when the background incidence of the disease is very low and any increased risk is modest. For commentators' views that the market and legal rules provide inadequate incentives to undertake the sorts of studies that provide information about agent-disease causation, see Mary L. Lyndon, *Information Economics and Chemical Toxicity: Designing Laws to Produce and Use Data*, 87 MICH. L. REV. 1795, 1810-1825 (1989) (public-good aspect of information); Wendy E. Wagner, *Choosing Ignorance in the Manufacture of Toxic Products*, 82 CORNELL L. REV. 773, 784-796 (1997) (identifying inadequacies in market, regulatory, and common-law incentives for adequate production of evidence of toxicity). The experience of defendants in the Bendectin and silicone-gel, breast-implant cases, in which plaintiffs managed substantial success until the litigation drove the development of science that tended to exonerate the agents, may provide incentives for some manufacturers for which these commentators fail to account.

While courts have permitted proof of general causation with something less than epidemiologic evidence, case reports--reports of an instance of disease in an individual following exposure to a given agent--have been found insufficient by themselves as proof of general causation. See Hollander v. Sandoz Pharms. Corp., 289 F.3d 1193, 1211 (10th Cir.2002); Siharath v. Sandoz Pharms. Corp., 131 F. Supp. 2d 1347, 1361-1362 (N.D.Ga.2001) (citing cases), aff'd, 295 F.3d 1194 (11th Cir.2002); Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH. L.J. 189, 216 (1992) (case reports and clusters of disease, while necessary, may only reflect coincidence due to random chance rather than a causal

relationship). But see Jennings v. Baxter Healthcare Corp., 14 P.3d 596, 607 (Or.2000) (suggesting that in an unusual case, with an especially powerful agent, case reports may be sufficient to establish causation). For a discussion of the other types of evidence bearing on general causation, see 2 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE § 27-1.0 to-1.3.2 (1997) (explaining animal toxicology, in vitro, and structure-activity studies); see also ERNEST HODGSON & PATRICIA LEVI, MODERN TOXICOLOGY (1987); MICHAEL A. KAMRIN, TOXICOLOGY: A PRIMER ON TOXICOLOGY PRINCIPLES AND APPLICATIONS (1988); Gerald W. Boston, *A Mass-Exposure Model of Toxic Causation: The Content of Scientific Proof and the Regulatory Experience*, 18 COLUM. J. ENVTL. L. 181, 218-231 (1993); Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 401 (2d ed. 2000); Jack L. Landau & W. Hugh O'Riordan, *Of Mice and Men: The Admissibility of Animal Studies to Prove Causation in Toxic Tort Litigation*, 25 IDAHO L. REV. 521 (1988-1989); Ellen K. Silbergeld, *The Role of Toxicology in Causation: A Scientific Perspective*, 1 CTS., HEALTH SCI. & LAW 374 (1991). For sources discussing the admissibility and sufficiency of toxicologic evidence, see FAIGMAN, supra, at § 27-1.1 n.11 (concluding "[i]t is impossible to reconcile all of the cases in this area").

The point about general causation existing in a background sense in all tort cases was drawn from Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107, 110 n.13 (2001); see also Richard W. Wright, *Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts*, 73 IOWA L. REV. 1001, 1046 (1988) ("Thus, to prove that a specific condition was a cause of a particular result, one obviously must establish ... that some credible causal generalization links conditions of that type to results of that type.").

Even when satisfactory evidence of general causation exists, such evidence generally supports proof of causation only for a specific disease. The vast majority of toxic agents cause a single disease or a series of biologically related diseases. (However, many different toxic agents may be combined in a single product, such as cigarettes.) When biological-mechanism evidence is available, it may permit an inference that a toxic agent caused a related disease. Otherwise, proof that an agent causes one disease is generally not probative of its capacity to cause other unrelated diseases. Thus, while there is substantial scientific evidence that asbestos causes lung cancer and mesothelioma, whether asbestos causes other cancers would require independent proof. Courts refusing to permit use of scientific studies that support general causation for diseases other than the one from which the plaintiff suffers unless there is evidence showing a common biological mechanism include Christophersen v. Allied-Signal Corp., 939 F.2d 1106, 1115-1116 (5th Cir.1991) (applying Texas law) (epidemiologic connection between heavy-metal agents and lung cancer cannot be used as evidence that same agents caused colon cancer); Cavallo v. Star Enters., 892 F. Supp. 756 (E.D. Va.1995), aff'd in part and rev'd in part, 100 F.3d 1150 (4th Cir.1996); Boyles v. Am. Cyanamid Co., 796 F. Supp. 704 (E.D.N.Y.1992). In Austin v. Kerr-McGee Ref. Corp., 25 S.W.3d 280, 290 (Tex.Ct.App.2000), the plaintiff sought to rely on studies showing that benzene caused one type of leukemia to prove that benzene caused a different type of leukemia in her decedent. Quite sensibly, the court insisted that before plaintiff could do so, she would have to submit evidence that both types of leukemia had a common biological mechanism of development.

For cases in which courts reached opposite conclusions on the value and adequacy of biological-mechanism evidence, compare Siharath v. Sandoz Pharms. Corp., 131 F. Supp. 2d 1347 (N.D.Ga.2001) (biological-mechanism evidence of effect of Parlodel, a drug that suppresses lactation, insufficient to permit plaintiff's expert witnesses to testify to general causation), aff'd, 295 F.3d 1194 (11th Cir.2002) with Tobin v. Astra Pharm. Prods., Inc., 993 F.2d 528 (6th Cir.1993) (applying Kentucky law) (plaintiff's expert relied predominantly on pathogenic evidence); Globetti v. Sandoz Pharms., Corp., 111 F. Supp. 2d 1174 (N.D.Ala.2000) (crediting expert witnesses who reasoned that because Parlodel is a vasoconstrictive agent it has the capacity to cause spasms that result in a heart attack); Stevens v. Sec'y of HHS, 2001 WL 387418, at *14 (Fed.Cl. March 30, 2001) (identifying infrequent instance when use of pathological-mechanism evidence is available and sufficiently probative to establish causation). However, scientists report that there is no methodology for assessing the strength or reliability of biological-mechanism evidence. It may vary from quite compelling to no more than hypothesis, with little supporting the latter other than some biologic knowledge and a fertile imagination. See generally Douglas L. Weed & Stephen D. Hursting, *Biologic Plausibility in Causal Inference: Current Methods and Practice*, 147 AM. J. EPIDEM. 415 (1998) (distinguishing between a plausible biological-mechanism hypothesis and biological-mechanism evidence based on research employing molecular biology and molecular epidemiology); Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH. L.J. 189, 230 (1992).

One final observation about the uncertainties of group observational studies and their use in civil litigation as proof of causation may assist those who do not regularly work in this area. The observational nature of epidemiologic studies virtually always results in concerns about the results being skewed by biases or unidentified confounders. Sampling error is also always possible in group studies, whether observational or experimental. Sometimes potential confounders can be identified and data gathered that permits analysis of whether confounding exists. Unidentified confounders, however, cannot be analyzed. Often potential biases can be identified, but assessing the extent to which they affected the study's outcome is problematical. Even sampling error, which is analyzed using quantitative statistical methods, only provides a range of outcomes (associations) that might have been produced by sampling error even if there is no association between the agent and disease. Thus, interpreting the results of epidemiologic studies requires informed judgment and is subject to uncertainty. Unfortunately, contending adversarial experts, because of the pressures of the adversarial system, rarely explore this uncertainty and provide the best, objective assessment of the scientific evidence. The extent of judgment involved in making causal assessments and range of uncertainty often involved augur for making that judgment with neutral, court-appointed experts, where feasible, whose expertise, judgment, and honest assessment of the degree of uncertainty involved can better be developed. An increasing number of judges, confronted with these issues, have chosen to employ court-appointed experts. See, e.g., Soldo v. Sandoz Pharms. Corp., 244 F. Supp. 2d 434 (W.D.Pa.2003); Miller v. Pfizer, Inc., 196 F. Supp.2d 1062, 1094 (D.Kan.2002); Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387 (D.Or.1996).

Complicating appropriate assessment of uncertainty and its implications for sufficiency of the evidence is that scientists generally do not think or express their judgments in probabilistic terms. While testimony as an expert in court is sometimes an exception, when scientists are asked to make an assessment of the degree of uncertainty based on existing evidence, they often have

difficulty responding. An example of this phenomenon occurred in an Institute of Medicine Committee that had been requested to examine the evidence bearing on a causal relationship between rubella vaccine and arthritis. The Committee report stated that the "evidence is consistent" with a causal relationship. Only after further inquiries were made did the Committee clarify that it meant that the Committee "favors acceptance of" a causal relationship. The court interpreting the latter categorization interpreted it to mean more likely than not. See Snyder v. Sect'y of HHS, 2002 WL 31965742 (Fed.Cl.2002).

(4) *Specific causation.* Applying the results of group studies to assess the probability of causation in an individual has become accepted by courts; this is especially true where, as is often the case, there is a lack of understanding about the other components of the casual chain necessary for a given disease. This acceptance has been necessitated by the legal requirement for proof of causation on an individual-plaintiff basis. Epidemiologists, however, do not seek to understand causation at the individual level and do not use incidence rates in group studies to determine the cause of an individual's disease. Epidemiologists may appreciate the conditions and caveats important to whether a study can appropriately be used to infer a probability of individual causation, but the process of doing so is not one that epidemiologists pursue outside the legal arena. See Michael D. Green et al., *Reference Guide on Epidemiology*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 381-382 (2d ed. 2000). Although addressed to ex ante risk assessments for individuals, the observations by two leading epidemiologists are equally applicable to ex post assessments of the probability of causation:

> We cannot measure the individual risk, and assigning the average value to everyone in the category reflects nothing more than our ignorance about the determinants of lung cancer that interact with cigarette smoke. It is apparent from epidemiologic data that some people can engage in chain smoking for many decades without developing lung cancer. Others are or will become primed by unknown circumstances and need only to add cigarette smoke to the nearly sufficient constellation of causes to initiate lung cancer. In our ignorance of these hidden causal components, the best we can do in assessing risk is to classify people according to measured causal risk indicators and then assign the average observed within a class to persons within the class.

KENNETH J. ROTHMAN & SANDER GREENLAND, MODERN EPIDEMIOLOGY 9 (2d ed. 1998).

Caution, however, is necessary in permitting or making inferences about specific causation based on an increased incidence found in a group study. One must appreciate that an association (increased incidence of disease among those exposed to the agent) found in a group study does not necessarily mean a causal relationship exists. Observational group studies are subject to a variety of errors--sampling error, bias, and confounding--and may, as a result, find associations that are spurious and not causal. Only after an evaluative judgment, based on the Hill criteria, that the association is likely to be causal rather than spurious, is a study valid evidence of general causation and specific causation. See Michael D. Green et al., *Reference Guide on Epidemiology, in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 333, 383-384 (2d ed. 2000). This judgment entails employing a number of unranked factors to decide if an inference of causation is appropriate. No scientific methodology exists for this process and, hence, reasonable scientists may, in some instances, come to different judgments about whether such an inference is appropriate. See generally Douglas L. Weed, *Epidemiologic Evidence and Causal Inference*, 14 HEMATOLOGY/ONCOLOGY CLINICS N. AM. 797

(2000).
Even if an association is judged to be causal, biases in a study may result in skewing the true magnitude of the risk. In addition, differences between those persons in the study group and the plaintiff to whom the study results are being applied must be considered. In some instances the results of a study on a different population may be inapplicable to others; for example, studies of risk factors for breast cancer in women would be inapplicable to men. In other cases, more subtle differences between the study population and the plaintiff may require consideration of whether the risk found in the study is equivalent for the plaintiff or, where information permits, should be adjusted. See In re Hanford Nuclear Reservation Litig., 1998 WL 775340, at *64-70 (E.D.Wash. Aug. 21, 1998) (addressing plaintiffs' expert's efforts to adjust probability of causation for individual plaintiffs based on individual factors, including genetic susceptibility), rev'd on other grounds, 292 F.3d 1124 (9th Cir.2002); Minn. Mining & Mfg. Co. v. Atterbury, 978 S.W.2d 183, 191 (Tex.Ct.App.1998). See generally David A. Freedman & Philip B. Stark, *The Swine Flu Vaccine and Guillain-Barré Syndrome: A Case Study in Relative Risk and Specific Causation*, 64 L. & CONTEMP. PROBS. 49 (2001) (criticizing the use of relative risk to determine probability of causation for individuals because of the risk of spurious associations and the extent of individual variation); David H. Kaye & David A. Freedman, *Reference Guide on Statistics*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 83, 96-97 & n.38 (2d ed. 2000) (explaining the problems of employing a study outcome to determine the probability of an individual's having contracted the disease from exposure to the agent because of variations in individuals that bear on the risk of a given individual contracting the disease); Mark Parascandola, *What is Wrong with the Probability of Causation*, 39 JURIMETRICS J. 29 (1998); Joseph Sanders, *Scientific Validity, Admissibility, and Mass Torts after* Daubert, 78 MINN. L. REV. 1387, 1401-1404 (1994); see also KENNETH J. ROTHMAN & SANDER GREENLAND, MODERN EPIDEMIOLOGY 9 (2d ed. 1998) ("As knowledge expands the risk estimates assigned to people will depart from average according to the presence or absence of other factors that affect the risk."); Gary E. Marchant, *Genetic Susceptibility and Biomarkers in Toxic Injury Litigation*, 41 JURIMETRICS J. 67, 67-68, 71-72, 90 (2000) (discussing role that knowledge about genetic contribution to disease might play in refining probability of causation based on epidemiologic studies of heterogenous populations).
The idea that a doubling of the incidence of disease in group studies is sufficient to support proof of specific causation is often accepted. Some courts have insisted on a doubling of disease as a minimum for proof of specific causation, while others have recognized that, if other known causes can be identified and eliminated, something less than a doubling would still support finding specific causation. See In re Hanford Nuclear Reservation Litig., 292 F.3d 1124, 1137 (9th Cir.2002) (applying Washington law) (recognizing the role of individual factors that may modify the probability of causation based on the relative risk); Allison v. McGhan Med. Corp., 184 F.3d 1300, 1315 n.16 (11th Cir.1999) (breast-implant case; relative risk of 2.0 is the threshold for an inference of specific causation; relative risk of 1.24 is insufficient); In re Joint E. & S. Dist. Asbestos Litig., 52 F.3d 1124 (2d Cir.1995) (applying New York law) (holding that plaintiff could provide sufficient evidence of causation without proving a doubling in the incidence of disease); Daubert v. Merrell Dow Pharms., Inc., 43 F.3d 1311, 1320 (9th Cir.1995) (applying California law) (requiring that plaintiff demonstrate a relative risk of 2); In re Joint E. & S. Dist. Asbestos Litig., 964 F.2d 92 (2d Cir.1992) (applying New York law) (relative risk less than 2.0 may still be sufficient to prove causation); DeLuca v. Merrell Dow Pharms., Inc., 911

F.2d 941, 958-959 (3d Cir.1990) (applying New Jersey law) (Bendectin allegedly caused limb-reduction birth defects); Magistrini v. One Hour Martinizing Dry Cleaning, 180 F. Supp. 2d 584, 606 (D.N.J.2002) ("[A] relative risk of 2.0 is not so much a password to a finding of causation as one piece of evidence, among others for the court to consider in determining whether an expert has employed a sound methodology in reaching his or her conclusion."); Miller v. Pfizer, Inc., 196 F. Supp. 2d 1062, 1079 (D.Kan.2002) (rejecting a threshold of 2.0 for the relative risk and recognizing that even a relative risk greater than 2.0 may be insufficient); In re Breast Implant Litig., 11 F. Supp. 2d 1217 (D.Colo.1998) (plaintiff must demonstrate more than a doubling of risk of disease by defendant's agent); Pick v. Am. Med. Sys., Inc., 958 F. Supp. 1151, 1160 (E.D.La.1997) (stating that a relative risk of 2 implies a 50% probability of specific causation, but acknowledging that a study with a lower relative risk is admissible, if not sufficient to support a verdict on causation); Sanderson v. Int'l Flavors & Fragrances, Inc., 950 F. Supp. 981, 1000 (C.D.Cal.1996) (relative risk of 2 is a threshold for plaintiff to prove specific causation); Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1403 (D.Or.1996) ("plaintiffs must be able to show a relative risk of greater than 2.0"); Manko v. United States, 636 F. Supp. 1419, 1434 (W.D.Mo.1986) (swine-flu vaccine allegedly caused Guillain-Barré syndrome), aff'd in part, 830 F.2d 831 (8th Cir.1987); Marder v. G.D. Searle & Co., 630 F. Supp. 1087, 1092 (D.Md.1986) (requiring a doubling of the relative risk in order to prove causation is more likely than not), aff'd without op. sub nom. Wheelahan v. G.D. Searle & Co., 814 F.2d 655 (4th Cir.1987); In re "Agent Orange" Prod. Liab. Litig., 597 F. Supp. 740, 835-837 (E.D.N.Y.1984) (Agent Orange allegedly caused a wide variety of diseases in Vietnam veterans and their offspring), aff'd, 818 F.2d 145 (2d Cir.1987); Cook v. United States, 545 F. Supp. 306, 308 (N.D.Cal.1982) (swine-flu vaccine allegedly caused Guillain-Barré syndrome); Stevens v. Sec'y of HHS, 2001 WL 387418, at *12-13 (Fed.Cl. March 30, 2001); Berry v. CSX Transp., Inc., 709 So. 2d 552, 569 n.13 (Fla.Dist.Ct.App.1998) (suggesting that a relative risk of less than 2 might be sufficient if other risk factors can be ruled out for the plaintiff); DePyper v. Navarro, 1995 WL 788828 (Mich.Cir. Ct. Nov. 27, 1995) (suggesting a relative risk of 2 is required for adequate proof of general causation); Landrigan v. Celotex Corp., 605 A.2d 1079, 1087 (N.J.1992) (relative risk greater than 2 "support[s] an inference that the exposure was the probable cause of the disease in a specific member of the exposed population"); Grassis v. Johns-Manville Corp., 591 A.2d 671, 675 (N.J.Super.Ct.App.Div.1991) ("The physician or other qualified expert may view the epidemiological studies and factor out other known risk factors such as family history, diet, alcohol consumption, smoking ... or other factors which might enhance the remaining risks, even though the risk in the study fell short of the 2.0 correlation."); Jones v. Owens-Corning Fiberglas Corp., 672 A.2d 230 (N.J.Super.Ct.App.Div.1996); McDaniel v. CSX Corp., 95 S.W.2d 257, 264 (Tenn.1997) (relative risk of 2 a factor to be considered but not required as a legal matter); Merrell Dow Pharms., Inc. v. Havner, 953 S.W.2d 706, 718 (Tex.1997) ("The use of scientifically reliable epidemiological studies and the requirement of more than a doubling of the risk strikes a balance between the needs of our legal system and the limits of science."). But cf. In re Fibreboard Corp., 893 F.2d 706, 711-712 (5th Cir.1990) (applying Texas law) (disapproving a trial in which several representative cases would be tried and the results extrapolated to a class of some 3000 asbestos victims, without consideration of any evidence about the individual victims; general causation, which ignores any proof specific to the individual plaintiff, could not substitute under Texas law for cause in fact.). Despite the considerable disagreement on whether a relative risk of two is required or merely a taking-off point for determining the sufficiency of

the evidence on specific causation, two commentators who surveyed the cases observe that "[t]here were no clear differences in outcomes as between federal and state courts." Russellyn S. Carruth & Bernard D. Goldstein, *Relative Risk Greater than Two in Proof of Causation in Toxic Tort Litigation*, 41 JURIMETRICS J. 195, 199 (2001).

The use of epidemiologic studies finding a doubling of disease in order to establish specific causation by a preponderance of the evidence rests on two important assumptions, one unarticulated. The first assumption is that the agent operates independently of other risk factors. When there is an interaction (i.e., the combined incidence of disease in those exposed to some other risk factor and to the agent is other than additive), it is not valid to use the increased incidence of disease due to one of the agents as a measure of the probability that an individual with the disease after exposure to both agents was caused by that agent. See Comment *c(5)*; Louis A. Cox, Jr., *Probability of Causation and the Attributable Risk*, 4 RISK ANALYSIS 221 (1984).

The unarticulated assumption involves the biology of disease development. The assumption is that the agent causes disease in individuals who would otherwise never have contracted it. An alternative possibility is that the agent accelerates the onset of disease that would otherwise have occurred in those individuals, albeit at a later time. See Sander Greenland & James M. Robins, *Conceptual Problems in the Definition and Interpretation of Attributable Fractions*, 128 AM. J. EPIDEMIOLOGY 1185 (1988); Sander Greenland & James M. Robins, *Epidemiology, Justice, and the Probability of Causation*, 40 JURIMETRICS 321 (2000). If an agent accelerates the onset of disease, rather than causing it in persons who would never otherwise have suffered from it, the excess incidence of disease in a group study will understate the proportion of persons whose disease was accelerated by the agent because incidence in group studies is based on the frequency of disease in a given period of time. See Greenland & Robins, supra; see also Ofer Shpilberg et al., *The Next Stage: Molecular Epidemiology*, 50 J. CLINICAL EPIDEMIOLOGY 633, 637 (1997) ("A 1.5- fold relative risk may be composed of a 5-fold risk in 10% of the population, and a 1.1-fold risk in the remaining 90%, or a 2-fold risk in 25% and a 1.1- fold for 75%, or a 1.5-fold risk for the entire population."). For studies whose results suggest acceleration, see Brad A. Racette, *Welding-Related Parkinsonism: Clinical Features, Treatments, and Pathophysiology*, 56 NEUROLOGY 8, 12 (2001) (stating that authors "believe that welding exposure acts as an accelerant to cause [Parkinson's Disease]"); James L. Gale et al., *Risk of Serious Acute Neurological Illness After Immunization with Diphtheria-Tetanus-Pertussis Vaccine A Population-Based Case-Control Study*, 271 JAMA 37, 41 (1994) (discussing finding in another study that risk of seizures following DPT vaccine administration were significantly higher within 6 days of administration; but, after 28 days, the incidence had dropped to normal).

Rarely will significant evidence bearing on the appropriate biological mechanism be available. If it were, permitting proof to contradict the assumption of nonacceleration would be attractive, save for one concern. If acceleration were able to be identified and proved, the measure of damages would also require reconsideration. Thus, accelerating the plaintiff's contraction of a chronic disease by two years would justify damages that are but a fraction of the damages appropriate if the agent caused a plaintiff to suffer from the disease for her remaining life. See Restatement Second, Torts § 924, Comment *e*; David A. Fischer, *Successive Causes and the Enigma of Duplicated Harm*, 66 TENN. L. REV. 1127 (1999). In addition, another impediment to employing small increases in risk as the basis for any kind of causal determination exists.

Group studies of toxic agents are observational, rather than experimental. The observational nature of these studies opens them to a variety of design and methodological errors that may produce spurious results. Random error is another potential cause of invalid results, regardless of whether the study is experimental or observational. Many scientists are leery of accepting a group study that finds an increased incidence of disease below a certain magnitude as demonstrating a true causal relationship. See Gary Taubes, *Epidemiology Faces Its Limits*, 269 SCI. 164 (1995) (reporting on a wide range of epidemiologists' expressing great skepticism of studies that find modest increases in incidence of disease unless replicated consistently in a number of independent studies); see also N. E. Breslow & N. E. Day, *Statistical Methods in Cancer Research*, *in* THE ANALYSIS OF CASE-CONTROL STUDIES 36 (IARC Pub. No. 32, Lyon, France 1980) ("[r]elative risks of less than 2.0 may readily reflect some unperceived bias or confounding factor"); David A. Freedman & Philip B. Stark, *The Swine Flu Vaccine and Guillain-barré Syndrome: A Case Study in Relative Risk and Specific Causation*, 64 LAW & CONTEMP. PROBS. 49, 60 (2001) ("If the relative risk is near 2.0, problems of bias and confounding in the underlying epidemiologic studies may be serious, perhaps intractable."); Samuel Shapiro, *Meta-analysis/Shmeta Analysis*, 140 AM. J. EPIDEM. 771, 772 (1994) (contending that meta-analysis can only minimize sampling error, leaving bias and confounding--"[r]elative risks of low magnitude (say, less than 2) are virtually beyond the resolving power of the epidemiologic microscope"). This concern is applicable to general causation rather than specific causation. The cautionary note is raised here because small relative risks are most often addressed in the specific-causation inquiry.

For an explanation of other assumptions necessary for the incidence rate to truly reflect the increased incidence of disease caused by exposure to the agent, see Jan Beyea & Sander Greenland, *The Importance of Specifying the Underlying Biologic Model in Estimating the Probability of Causation*, 76 HEALTH PHYSICS 269, 271-272 (1999); Sander Greenland & James M. Robins, *Epidemiology, Justice, and the Probability of Causation*, 40 JURIMETRICS 321, 332-333 (2000) (explaining assumptions that: 1) agent does not cause other diseases that might be a competing cause of death; and 2) that doses of the agent do not prevent or delay the onset of diseases among some of those exposed).

A technique sometimes available to assist in proof of specific causation is a differential diagnosis. The idea is that a cause may be identified by eliminating the possibility that other known and alternative causes were responsible for the outcome. Many courts have endorsed such use. See, e.g., Siharath v. Sandoz Pharms. Corp., 131 F. Supp. 2d 1347 (N.D.Ga.2001), aff'd, 295 F.3d 1194 (11th Cir.2002); Globetti v. Sandoz Pharms., Corp., 111 F. Supp. 1174 (N.D.Ala.2000); Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1413 (D.Or.1996); John's Heating Serv. v. Lamb, 46 P.3d 1024, 1035 (Alaska 2002); U.S. Sugar Corp. v. Henson, 823 So. 2d 104 (Fla.2002); Schafersman v. Agland Coop., 631 N.W.2d 862, 871 (Neb.2001); Alder v. Bayer Corp., 61 P.3d 1068, 1084-1085 (Utah 2002); Easum v. Miller, 92 P.3d 794 (Wyo.2004); see also Martin v. Shell Oil Co., 180 F. Supp. 2d 313, 318- 319 (D.Conn.2002) (plaintiff's expert not required to perform a differential diagnosis where other evidence of specific causation is employed). For a thorough discussion of the cases and the issues posed by such evidence, see Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107 (2001).

More generally, the methodology of identifying a cause by eliminating other known causes of the

outcome is widely employed in a variety of investigative fields. See, e.g., Baker Valley Lumber, Inc. v. Ingersoll Rand Co., 813 A.2d 409 (N.H. Dec. 12, 2002) (fire investigators attempting to determine the cause of a sawmill fire); Morton M. Hunt, *The Case of Flight 320*, THE NEW YORKER, Apr. 30, 1960, at 119 (explaining the method by which investigators for the predecessor to the National Transportation Safety Board attempt to determine the cause of an airplane crash). In Stubbs v. City of Rochester, 124 N.E. 137 (N.Y.1919), one of the classic cases on proof of causation, the court sanctioned plaintiff's effort to prove that the defendant's intermingling of sanitary drinking water and unsanitary water was the cause of his typhus by eliminating many (but not all) of the other known causes of typhus.

In the medical profession, "differential diagnoses" are frequently employed to determine the patient's disease rather than its cause. When the cause of a disease, such as cancer, is not of clinical significance, physicians do not attempt to determine it. See Roach v. PPG Indus., Inc., ___ S.W.2d ___ (Ark.Ct.App.2004) (treating physician's opinion on cause of patient's cancer held inadmissible: "[The expert] was clearly more concerned with identifying and treating the decedent's condition than he was with identifying the specific substance that caused his condition. He arrived at his opinion about benzene more as an afterthought, in an ad hoc manner."). Only when the cause may have some continuing effect on the patient's health, as when a rash may be the result of a continuing occupational exposure, do physicians attempt to determine the cause of the patient's problems. For an explanation of the difference between the medical and legal communities in usage of the terms "differential diagnosis" and "differential etiology," see Mary Sue Henifin et al., *Reference Guide on Medical Testimony*, *in* FEDERAL JUDICIAL CENTER, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 439, 443-444 (2d ed. 2000); Edward J. Imwinkelreid, *The Admissibility and Legal Sufficiency of Testimony about Differential Diagnosis (Etiology): of Under--and Over--Estimations*, 56 BAYLOR L. REV. 391, 402-403 (2004); see also Turner v. Iowa Fire Equipment Co., 229 F.3d 1202, 1208 (8th Cir.2000) (distinguishing between differential diagnosis conducted for the purpose of identifying the disease from which the patient suffers and one attempting to determine the cause of the disease); Kannankeril v. Terminix Int'l, Inc., 128 F.3d 802, 807 (3d Cir.1997) (differential diagnosis is "the determination of which of two or more diseases with similar symptoms is the one from which the patient is suffering, by a systematic comparison and contrasting of the clinical findings," quoting STEDMAN'S MEDICAL DICTIONARY 428 (25th ed. 1990)); Hodgdon v. Frisbie Mem'l Hosp., 786 A.2d 859 (N.H.2001) (employing differential diagnosis to describe the process of diagnosing patient's disease based on clinical symptoms); Yacoub v. Lehigh Valley Med. Assocs., P.C., 805 A.2d 579 (Pa.Super.Ct.2002); MOSBY'S MEDICAL & NURSING DICTIONARY 347 (2d ed. Walter D. Glanze et al. eds., 1986) (defining differential diagnosis as "the distinguishing between two or more diseases with similar symptoms by systematically comparing their signs and symptoms"). For an example of the customary use of the phrase "differential diagnosis" in the clinical medical community, see W. Scott Richardson, *Users' Guide to the Medical Literature: XV. How to Use an Article About Disease Probability for Differential Diagnosis*, 281 JAMA 1214 (1999).

The validity of a differential diagnosis depends on a substantial proportion of the independent causes of the disease being known. This means that each of those causes operates independently of the others and (along with background causes) causes some percentage of all cases of the disease in question. When a number of the causes are known and the plaintiff can be evaluated for the existence of such causes, the probability of an agent causing the plaintiff's disease can be

evaluated. A differential diagnosis is of limited utility when a substantial portion of the incidence of a disease is of unknown etiology. See Susan R. Poulter, *Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?*, 7 HIGH TECH. L.J. 189, 233 (1992); Coastal Tankships, U.S.A., Inc. v. Anderson, 87 S.W.3d 591, 614-615 (Tex.Ct.App.2002) (Brister, J., concurring). An example of a disease for which most of the causes are unknown is birth defects. Estimates are that some 2/3 of all birth defects are due to unknown causes. See Robert L. Brent, *The Complexities of Solving the Problem of Human Malformations*, 13 CLINICS IN PERINATOLOGY 491, 493 (1986) (citing various estimates of the proportion of birth defects due to unknown causes).

An excellent explanation for why differential diagnoses generally are inadequate without further proof of general causation was provided in Cavallo v. Star Enter., 892 F. Supp. 756 (E.D.Va.1995), aff'd in relevant part, 100 F.3d 1150 (4th Cir.1996):

> The process of differential diagnosis is undoubtedly important to the question of "specific causation". If other possible causes of an injury cannot be ruled out, or at least the probability of their contribution to causation minimized, then the "more likely than not" threshold for proving causation may not be met. But, it is also important to recognize that a fundamental assumption underlying this method is that the final, suspected "cause" remaining after this process of elimination must actually be capable of causing the injury. That is, the expert must "rule in" the suspected cause as well as "rule out" other possible causes. And, of course, expert opinion on this issue of "general causation" must be derived from a scientifically valid methodology.

Id. at 771 (footnote omitted); see also Hollander v. Sandoz Pharms. Corp., 289 F.3d 1193, 1210-1211 (10th Cir.2002) (district court did not abuse its discretion in excluding expert testimony based on differential diagnosis without other adequate evidence of general causation); Meister v. Med. Eng'g Corp., 267 F.3d 1123 (D.C.Cir.2001); Lennon v. Norfolk and W. Ry. Co., 123 F. Supp. 2d 1143, 1153-1154 (N.D.Ind.2000); Stevens v. Sec'y of HHS, 2001 WL 387418, at *20 (Fed.Cl. March 30, 2001); E.I. du Pont de Nemours & Co. v. Robinson, 923 S.W.2d 549 (Tex.1995); Coastal Tankships, U.S.A., Inc. v. Anderson, 87 S.W.3d 591 (Tex.Ct.App.2002); see generally Joseph Sanders & Julie Machal-Fulks, *The Admissibility of Differential Diagnosis Testimony to Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 LAW & CONTEMP. PROBS. 107, 122-125 (2001) (discussing cases rejecting differential diagnoses in the absence of other proof of general causation and contrary cases).

Despite the *Cavallo* court's appropriate insistence on "ruling in" with evidence of general causation, Zuchowicz v. United States, 140 F.3d 381 (2d Cir.1998) (Federal Tort Claims Act case adopting Connecticut law), provides the unusual circumstances for an exception. Plaintiff was exposed to a substantial overdose of a drug due to negligence by the prescribing physician and developed primary pulmonary hypertension (PPH), which resulted in her death. No studies had been conducted of doses of the magnitude taken by the deceased, and there were very few humans ever exposed to doses that high, thereby explaining why human studies did not exist. (The court did not mention the existence of, or reason for the absence of, animal toxicology studies.) Plaintiff's experts provided an apparently plausible explanation of the biological mechanism by which the overdose caused PPH, backed at least with regard to some aspects by a variety of studies. Plaintiff's experts also ruled out, through a differential diagnosis, some of the other causes of PPH, including other drugs. The decedent exhibited symptoms of drug-induced PPH shortly after her overdose and the disease progressed consistently with other drug-induced

cases of PPH. Based on this evidence, the court of appeals affirmed the district court's decision admitting the expert testimony and refused to disturb the finding of causation by the court, sitting as factfinder. See also Westberry v. Gislaved Gummi AB, 178 F.2d 257 (4th Cir.1999) (acute response, differential diagnosis ruled out other known causes of disease, dechallenge, rechallenge tests by expert that were consistent with exposure to defendant's agent causing disease, and absence of epidemiologic or toxicologic studies; holding that expert's testimony on causation was properly admitted). As a prominent medical clinician and former editor of the New England Journal of Medicine observed, "Unfortunately no set formula or algorithm exists for deciding whether a human illness or condition is the consequence of a given exposure to a drug, chemical or some other agent." Jerome P. Kassirer, Joint Discussion of Science, Technology, and Law Panel and American Law Institute: Restatement of Torts 12 (Jan. 21, 2003), at http://www7.nationalacademies.org/stl/index.html. This comment could encompass the variety of circumstances that may exist in a toxic-substances case affecting the available and appropriate proof, the lack of any methodology on employing the Hill criteria to determine whether an inference of causation is appropriate based on a group study that finds an association, the absence of any scientific methodology or guidelines for determining specific causation from group studies, or the lack of any established methodology for clinical judgments of causality.

(5) *Multiple exposures and synergistic interactions*. The discussion of multiple exposures and synergistic interactions in this Comment assumes a disease for which the severity of the disease is not dose dependent. Asbestosis is an example of a disease whose severity *is* dose dependent: the severity of the disease in an individual is a function of that individual's exposure level. For such diseases, each additional unit of exposure causes some marginal additional harm. By contrast, the severity of many cancers is assumed not to be dependent on the dose to which the individual was exposed. For these non-dose-dependent diseases, each exposure is a cause of the disease. See § 27, Comment *g*. It is the latter class of diseases (or ones that are treated as such) that is addressed in this Comment.

In this multiple-exposure situation, courts have adopted a rule that each nontrivial exposure to a toxic agent may be found by the trier of fact to be a factual cause of plaintiff's disease. See § 27, Comment *g*, and Reporters' Note thereto. Alternatively, especially when plaintiff has been exposed to the toxic agent of multiple defendants, as is frequently the case with asbestos, plaintiffs would be presented with proving which exposure initiated the disease. If a court adopted the latter requirement, it would then be confronted with the propriety and application of alternative liability, see § 28(b), and shifting the burden of proof to defendants on the question of factual causation. See Rutherford v. Owens-Ill., Inc., 941 P.2d 1203, 1215-1220 (Cal.1997).

The difference between these two proof requirements results from different assumptions about the biology of disease development. The asbestos rule (a "threshold rule") rests on an assumption that each dose of asbestos contributes to a threshold dose above which disease is caused in the individual exposed. See Eagle-Picher Indus., Inc. v. Balbos, 604 A.2d 445 (Md.Ct.App.1992) (expert testifying to belief in an "undefined 'threshold' of asbestos exposure" required before disease would occur). Alternatively, each dose of a carcinogen may pose an independent and distinct, albeit small, risk of causing cancer in the exposed individual. Indeed, the "one-hit" model of carcinogenesis is consistent with this no-threshold hypothesis. See Committee on Risk Assessment of Hazardous Air Pollutants, National Research Council, Science and Judgment in Risk Assessment 123-124 (1994); Committee on the Institutional Means for Assessment of Risks to Public Health, National Research Council, Risk Assessment in the Federal Government:

Managing the Process 19-20 (1983); Joseph V. Rodricks & Susan H. Rieth, *Toxicological Risk Assessment in the Courtroom: Are Available Methodologies Suitable for Evaluating Toxic Tort and Product Liability Claims?*, 27 Reg. Toxicology & Pharmacology 21, 23-24 (1997).

The court in Rutherford v. Owens-Illinois, Inc., 941 P.2d 1203, 1218 (Cal.1997), recognized these two different causal models to explain the role of asbestos in causing asbestotic malignancies. Because of the impossibility of proof that would be posed if the second model were assumed, the court adhered to a threshold rule model, only requiring that plaintiff's exposure to defendant's asbestos was a "substantial factor in contributing to the aggregate dose of asbestos" to which the plaintiff was exposed. Id.

In Manguno v. Babcock & Wilcox, 961 F.2d 533 (5th Cir.1992) (applying Louisiana law), plaintiffs were smokers who were occupationally exposed to asbestos and suffered from lung cancer. They sued several asbestos manufacturers who argued that plaintiffs could not recover without proving that their lung cancer would not have occurred but for the exposure to asbestos. None of plaintiffs' experts testified to that, instead characterizing the asbestos exposure as a significant contributing factor, while recognizing the causal role of smoking as well. The trial court instructed the jury as requested by defendants, and the court of appeals reversed. Initially, the court of appeals was confronted with the plaintiffs' argument that but-for causation is not required in a multiple sufficient causal case. See § 27. Defendants countered that the case did not present a multiple sufficient causal situation and therefore but-for cause was appropriate. The court of appeals recognized (or assumed) that the case did not present multiple sufficient causes. Nevertheless, the court denied that multiple sufficient causes was the only situation in which but-for cause was relaxed. The court cited multiple asbestos-exposure cases, see § 27, Comment *g*, and held that the but-for causal instruction was erroneous. Thus, the court of appeals adapted the threshold rule to the case of multiple exposures to different toxic agents, each of which is a risk factor for plaintiff's disease.

When synergies exist, the question of how to allocate the probability attributable to the synergistic (supra-additive) effect of the combined agents (in addition to the probability due to each agent alone) requires normative judgment and cannot be calculated simply by mathematical technique. See Grahn v. Dillingham Constr., Inc., 2004 WL 2075570 (Cal.Ct.App. Sept. 17, 2004) (affirming exclusion of expert witness's testimony that plaintiff's cancer was more probably due to smoking than exposure to asbestos and that probability could be readily determined with "just math."). The expert's testimony in *Grahn* not only ignored the question of how to allocate the probability due to the synergistic effect, it also assumed that cigarette smoking and asbestos exposure operate independently of each other in causing cancer. See Illustrations 2-3.

The discussion of synergy and Illustrations 4 and 5 are based on a number of cases in which plaintiffs were exposed to both asbestos and cigarette smoke. In Dafler v. Raymark Indus., Inc., 611 A.2d 136 (N.J.Super.Ct.App.Div.1992), aff'd, 622 A.2d 1305 (N.J.1993), the court permitted apportionment of liability without a determination that the plaintiff's smoking constituted contributory negligence (or that the manufacturer of the cigarettes was liable for the plaintiff's harm). The *Dafler* court relied on § 433A, Comment *a*, of the Second Restatement of Torts, which provides for causal apportionment. That reliance may not be justified, however. Comment *a* states that its provisions "apply also where one of the causes in question is the conduct of the plaintiff himself, whether it be negligent or innocent." That statement is best read as inapplicable to a harm, the entirety of which was caused by both innocent-plaintiff conduct and tortious

conduct by a defendant. Only when the innocent conduct and the tortious conduct each cause less than the entirety of the harm is causal apportionment appropriate. While Subsection (1)(b) of § 433A suggests that causal apportionment might be appropriate even when the innocent conduct, along with the tortious conduct, was a cause of the entirety of the harm, if "a reasonable basis for determining the contribution of each cause to a single harm" exists, all of the innocent-cause discussion and Illustrations are consistent with the innocent cause causing less than the entirety of damages. Apportionment is then based on what harm was caused by each of the defendants and the innocent plaintiff. Any contrary reading would run afoul of long-time and well-established rules of causation, which merely require that tortious conduct be *a* cause of harm for liability, regardless of any other causes that may exist. Indeed, the Second Restatement recognizes these principles and evidences inconsistency in Subsection (2) of § 433A and Comment *i*, which observes that some harms cannot be apportioned and includes as examples death, a broken limb, destruction of real property, and an Illustration involving the death of cattle. Comment *i* also recognizes that the existence of other innocent causes in the causal chain producing these outcomes does not change the result. The inconsistency between Subsection (1)(b) of § 433A and Comment *i* is best demonstrated by two Illustrations, virtually identical, that involve two defendants, each of whom pollutes a stream with oil. Illustration 5 permits apportionment in a nuisance case based on the proportion of oil provided (70/30) by each of the defendants. Illustration 15 bars apportionment for the death of plaintiff's cattle, poisoned by drinking oil, without any mention of evidence of the respective contribution of oil by defendants. Apportionment based on causation appears more justified in Illustration 15, as the oil of each defendant could have caused the discrete deaths of some of the plaintiff's cattle, although that evidence is unlikely to be available. The exception for "exceptional cases" in Comment *h*, including instances when one defendant is insolvent, is also inconsistent with Subsection (1) of § 433A. See Gerald W. Boston, *Toxic Apportionment: A Causation and Risk Contribution Model*, 25 ENVTL. L. 549, 568-572 (1995) (criticizing some "problems" with § 433A of the Second Restatement).

At the time the Second Restatement was drafted and approved, there was no apportionment based on comparative responsibility between a plaintiff and defendant; multiple defendants who were liable for a single harm were jointly and severally liable; and apportionment among them was pro rata. The widespread adoption of comparative responsibility and modification of joint and several liability have changed those rules. Section 433A has been superseded by Restatement Third, Torts: Apportionment of Liability § 26.

The apportionment sanctioned by *Dafler* was based roughly on the ratio of increased risks posed by plaintiff's exposure to asbestos and to cigarette smoke. This evidence was provided by plaintiff's expert witnesses, one of whom testified that it was impossible to apportion plaintiff's disease based on causation, and the other of whom testified he could not apportion based on "responsibility." Defendant's expert provided no basis for apportioning, opining that plaintiff's disease was due solely to smoking. Nevertheless, the court found that a reasonable basis for apportioning liability existed and affirmed the trial court's decision to submit this question to the jury. Other courts have held that similar evidence is insufficient to permit apportionment based on causation. See Borman v. Raymark Indus., Inc., 960 F.2d 327, 334- 335 (3d Cir.1992) (applying Pennsylvania law) (denying apportionment based on § 433A of the Restatement Second of Torts despite relative-risk evidence similar to that presented in *Dafler*); Martin v. Owens-Corning Fiberglas Corp., 528 A.2d 947, 949 (Pa.1987). The *Dafler* court's reliance on an

earlier New Jersey decision holding that a plaintiff's emotional distress due to an abortion could be causally apportioned among her physician, who was negligent in an earlier sterilization procedure, a negligent driver, who caused an accident that resulted in the plaintiff who did not know she was pregnant at the time, being x-rayed, and the treating physician, who x-rayed her, reveals that the New Jersey courts do not make the same sharp distinction between apportionment on the basis of comparative responsibility and on the basis of causation provided by the Apportionment of Liability Restatement. See Bendar v. Rosen, 588 A.2d 1264 (N.J.Super.Ct.App.Div.1991) (invoking apportionment based on comparative responsibility and causation without acknowledging the difference).

The overwhelming number of courts that permit apportionment between smoking and asbestos exposure base it on comparative-responsibility principles, rather than causation, and therefore require a finding of contributory negligence by the plaintiff in smoking (liability of a person who manufactured or sold the cigarettes). See Ingram v. ACandS, Inc., 977 F.2d 1332, 1340-1341, 1342 (9th Cir.1992) (applying Oregon law) (rejecting apportionment based on causation but upholding comparative-responsibility apportionment both among asbestos defendants and between defendants and plaintiff who smoked); Zarow-Smith v. N. J. Transit Rail Operations, 953 F. Supp. 581 (D.N.J.1997) (upholding comparative-responsibility apportionment to plaintiff based on smoking in FELA case); Fulgium v. Armstrong World Indus., Inc., 645 F. Supp. 761, 763 (W.D.La.1986) (evidence of plaintiff's smoking admissible for purposes of comparative-responsibility apportionment); Richards v. Owens-Ill., Inc., 928 P.2d 1181 (Cal.1997) (statutory immunity of tobacco suppliers prevented apportionment of comparative responsibility to them); Champagne v. Raybestos-Manhattan, Inc., 562 A.2d 1100, 1118 (Conn.1989) (permitting plaintiff's smoking to be the basis for comparative-responsibility apportionment, citing Brisboy v. Fibreboard Corp., infra); In re Asbestos Litig. Pusey Trial Group, 669 A.2d 109, 111-113 (Del.1995) (trial court erred in permitting apportionment without instructing on and requiring jury to find that plaintiff's smoking constituted comparative fault); Hao v. Owens-Ill., Inc., 738 P.2d 416 (Haw.1987) (same); Owens Corning Fiberglas Corp. v. Parrish, 58 S.W.3d 467 (Ky.2001) (upholding comparative-responsibility assignment to plaintiffs based on their smoking); Brisboy v. Fibreboard Corp., 418 N.W.2d 650, 655-656 (Mich.1988); cf. Jones v. Owens-Corning Fiberglas Corp., 69 F.3d 712 (4th Cir.1995) (applying North Carolina law) (contributory fault by plaintiff in smoking cigarettes can be asserted by asbestos defendants); Gideon v. Johns-Manville Sales Corp., 761 F.2d 1129, 1138-1140 (5th Cir.1985) (applying Texas law) (evidence of asbestosis victim's smoking admissible for purposes of defendant's claim of mitigation of damages and for jury to decide if future risk of cancer was caused by smoking, asbestos exposure, or both).

Professor Boston, in a thorough and careful article, advocates apportionment based on risk contribution similar to *Dafler*. Gerald W. Boston, *Toxic Apportionment: A Causation and Risk Contribution Model*, 25 ENVTL. L. 549, 572-591 (1995). He does so in apportioning liability among multiple defendants because of his concern about the unfairness of holding a defendant jointly and severally liable when there are other defendants who contributed to the risk of disease who are insolvent. Yet apportionment can be on the basis of comparative responsibility, rather than risk apportionment, and joint and several liability can be modified if a jurisdiction wishes. See Restatement Third, Torts: Apportionment of Liability § 17, Reporters' Note to Comment *a* (detailing jurisdictions that have modified joint and several liability). Professor Boston favors risk-contribution apportionment when a plaintiff's smoking concurs with asbestos exposure in

order to further incentives for persons to assume responsibility for safer lifestyles. Yet tort law does not impose liability (or reduce recovery) to provide deterrence unless the person has engaged in tortious conduct. Absent a finding of contributory negligence by the plaintiff, there is no basis for apportioning liability on the basis of causation to the plaintiff. Taken to its logical extreme, Professor Boston's risk-contribution apportionment approach would permit apportionment of liability to a plaintiff whose genotype contributed to the risk, once that information is available, or even to exposure to sunlight or other environmental factors that also are risk factors for disease. Well-settled law does not permit apportionment of liability or causal contribution to a preexisting condition. See U. S. Fid. & Guar. Co. v. United States, 152 F.2d 46, 49 (2d Cir.1945); Buchalski v. Universal Marine Corp., 393 F. Supp. 246, 248 (W.D.Wash.1975).

Another basis for apportioning liability in cases like those discussed above is on the basis of damages. Thus, an asbestos manufacturer might claim that, although plaintiff's lung cancer was caused by exposure to asbestos, plaintiff's smoking would have caused death at an early age, and therefore the damages should be reduced. See Restatement Second, Torts § 924, Comment *e*. Because questions of damages are beyond the scope of this Restatement and because of the absence of any cases addressing this matter, this Restatement does not address this means of apportionment.

For a study of asbestos workers that finds increased risks relating to asbestos exposure, smoking, and relative risks similar in magnitude to those in Illustrations 4 and 5, see Piero Mustacchi, *Lung Cancer Latency and Asbestos Liability*, 17 J. LEGAL MED. 277 (1996).

Comment d. Burden of proof on magnitude of divisible harm

(1) Apportioning harm caused by the legally culpable conduct of multiple parties. This Comment and much of this Reporters' Note is fashioned from treatment in Restatement Third, Torts: Apportionment of Liability § 26, Comment *h*.

An example of the turn-of-the-century attitude toward plaintiffs and their burden of proof to show the magnitude of injury caused by the defendant focused on the potential unfairness to defendants:

> [I]f the rule was as contended ... one who has caused an offensive odor by the slaughtering of one animal near a slaughter-house, where great numbers are killed and an actionable nuisance created, would be held for the entire damage done; where the smoke of one chimney of a residence near a brick kiln, which has become a nuisance united with that of the kiln, the owner of the residence would be liable for all the damages caused by the nuisance....

Swain v. Tenn. Copper Co., 78 S.W. 93, 95 (Tenn.1903). Another court that required plaintiff to prove the extent of harm caused by each tortfeasor is Sun Oil Co. v. Robicheaux, 23 S.W.2d 713, 715 (Tex.App.1930) (tortfeasor liable only for the "damages, which directly and proximately result from his own act, and the fact that it may be difficult to define the damages caused by the wrongful act of each person who independently contributed to the final result does not affect the rule").

Avoiding the unfairness identified in *Swain* resulted in corresponding unfairness to plaintiffs. This unfairness was expressed well by the Michigan Supreme Court:

> When we impose upon an injured plaintiff the necessity of proving which impact did which harm in a chain collision situation, what we are actually expressing is a judicial policy that it is better that a plaintiff, injured through no fault of his own, take nothing, than a tortfeasor pay more than his theoretical share of the damages accruing out of a confused situation which

his wrong has helped to create.

Maddux v. Donaldson, 108 N.W.2d 33, 35 (Mich.1961).

Many courts responded similarly, as did the Texas Supreme Court, when it overruled *Sun Oil*. See Landers v. Tex. Salt Water Disposal Co., 248 S.W.2d 731 (Tex.1952); see also Woodward v. Blythe, 462 S.W.2d 205, 208-209 (Ark.1971); Vereb v. Markowitz, 108 A.2d 774 (Pa.1954). Defendants who caused part of an injury were held jointly and severally liable for the entire injury unless a defendant could prove the magnitude of the damages caused by that defendant. See 3 FOWLER V. HARPER, FLEMING JAMES, JR. & OSCAR S. GRAY, THE LAW OF TORTS § 10.1, at 4-5 (2d ed. 1986); Restatement Second, Torts § 433B(2).

Some courts have responded to the unfairness of imposing a rigorous burden of proof on plaintiffs by relaxing the burden of production. The rationale is explained well in Cal. Orange Co. v. Riverside Portland Cement Co., 195 P. 694 (Cal.Ct.App.1920):

> In determining the amount of damages that should be assessed against this defendant, the trial court was at liberty to estimate as best it could, from the evidence before it, how much of the total damage, caused by the operations of the two cement companies, was occasioned by defendant's plant, and, in doing so, might measure with a liberal hand the amount of damage caused by defendant's mill.... Though in cases of this sort entire accuracy is impossible, and the difficulty of accurately proportioning and assessing the damage done by defendant's mill is great, still that difficulty would have been avoided had defendant but taken care that no occasion should arise requiring such proportioning and assessing of the whole damages.

See also Schultz v. Pivar, 88 A.2d 74 (Pa.1952). For an excellent history of these doctrines, see Gerald Boston, *Apportionment of Harm in Tort Law: A Proposed Restatement*, 21 U. DAYTON L. REV.. 267, 270-274 (1996). An opinion discussing the burden of proof on the magnitude of harm in a crashworthiness case and revealing the trend toward only requiring plaintiff to prove some enhancement due to the product defect rather than the amount of enhancement is Stecher v. Ford Motor Co., 779 A.2d 491 (Pa.Super.Ct.2001); see also Restatement Third, Torts: Products Liability § 16. For discussion of other areas of tort law in which courts have adopted a relaxed burden of proof with regard to the magnitude of damages, see Pati Jo Pofahl, Smith v. Superior Court: *A New Tort of Intentional Spoliation of Evidence*, 69 MINN. L. REV. 961, 969-973 (1985).

(2) Apportioning harm between tortious conduct and innocent causes or nonparty actors. Courts are split on whether to impose the burden of proof with regard to the magnitude of the aggravated harm on plaintiff or defendant. Compare LaMoureaux v. Totem Ocean Trailer Express, Inc., 632 P.2d 539, 544-545 (Alaska 1981); Blatz v. Allina Health Sys., 622 N.W.2d 376 (Minn.Ct.App.2001); Reichert v. Vegholm, 840 A.2d 942 (N.J.Super.Ct.App.Div.2004); Blanks v. Murphy, 632 A.2d 1264 (N.J.Super.Ct.App.Div.1993); Morris v. Rogers, 456 P.2d 863 (N.M.1969), with Newbury v. Vogel, 379 P.2d 811 (Colo.1963); Wise v. Carter, 119 So. 2d 40 (Fla.Dist.Ct.App.1960); Kawamoto v. Yasutake, 410 P.2d 976 (Haw.1966); Browning v. Ringel, 995 P.2d 351 (Idaho 2000); Lovely v. Allstate Ins. Co., 658 A.2d 1091 (Me.1995); McNabb v. Green Real Estate Co., 233 N.W.2d 811 (Mich.Ct.App.1975); Callihan v. Burlington N. Inc., 654 P.2d 972 (Mont.1982); David v. DeLeon, 547 N.W.2d 726 (Neb.1996); Shippen v. Parrott, 553 N.W.2d 503 (S.D.1996); Haws v. Bullock, 592 S.W.2d 588, 591 (Tenn.Ct.App.1979); Tingley v. Christensen, 987 P.2d 588, 592 (Utah 1999) ("[I]f a jury can find a reasonable basis for apportioning the damages between a preexisting condition and a subsequent tort, it should do so; however, if the jury finds it impossible to apportion damages, it should find that the tortfeasor is

liable for the entire amount of the damages."); Cox v. Spangler, 5 P.3d 1265 (Wash.2000); Bigley v. Craven, 769 P.2d 892 (Wyo.1989). In some cases, courts functionally place the burden of proof on defendant when the extent of aggravation is uncertain by declaring the plaintiff's harm to be indivisible and therefore not subject to apportionment.

An excellent illustration of the ambivalence that exists about where to place the burden of proof in cases such as these is revealed in a passage in Washewich v. LeFave, 248 So. 2d 670, 672-673 (Fla.Dist.Ct.App.1971):

> On the basis of the foregoing authority, it is our opinion that where the evidence reveals two successive accidents, and the defendant is only responsible for the second accident, the burden is on the plaintiff to prove to the extent reasonably possible what injuries were proximately caused by each of the two accidents. The jury should be instructed to make an apportionment of the damages as between the two accidents insofar as it may be reasonably possible to do so, but if an apportionment is impossible, the jury may be authorized to charge the defendant with all damages flowing from the entire injury.

Comment e. Burden shifting when plaintiff cannot reasonably demonstrate which of several tortfeasors caused the harm. There has been substantial support for the principle contained in § 433B(3) of the Second Restatement of Torts. The advent of toxic-substances litigation, especially asbestos and DES, both of which involved products with long latency periods and a large number of manufacturers, confronted courts with determining the limits of alternative liability and its propriety in mass-victim, multiple-manufacturer cases, such as DES and asbestos.

For cases affirming the principle of alternative liability since the Second Restatement, see Bowman v. Redding & Co., 449 F.2d 956 (D.C.Cir.1971) (applying District of Columbia law); Zands v. Nelson, 797 F. Supp. 805 (S.D.Cal.1992) (federal law under Resources Conservation and Recovery Act); McElhaney v. Eli Lilly & Co., 564 F. Supp. 265, 269 (D.S.D.1983) (predicting that the South Dakota Supreme Court would adopt § 433B(3)); Wysocki v. Reed, 583 N.E.2d 1139 (Ill.App.Ct.1991) (relying on alternative liability as basis for permitting legal-malpractice claims against attorney who did not pursue plaintiff's claim because of inability to identify tortfeasor); Abel v. Eli Lilly & Co., 343 N.W.2d 164, 172 (Mich.1984); McMillan v. Mahoney, 393 S.E.2d 298 (N.C.Ct.App.1990); NOPCO Chem. Div. v. Blaw-Knox Co., 281 A.2d 793 (N.J.1971) (consecutive bailees of commercial machine damaged while it was in the possession of one of the bailees; burden of production placed on defendants, with burden of persuasion remaining with plaintiff); Minnich v. Ashland Oil Co., 473 N.E.2d 1199 (Ohio 1984); Hood v. Hagler, 606 P.2d 548, 553-554 (Okla.1979); Snoparsky v. Baer, 266 A.2d 707 (Pa.1970); Martin v. Abbott Labs., 689 P.2d 368, 375 (Wash.1984) ("Alternate liability has been recognized, but not as of yet applied in this state."); Fiumefreddo v. McLean, 496 N.W.2d 226 (Wis.Ct.App.1993).

The Canada Supreme Court adopted alternative liability in Lewis v. Cook, [1951] S.C.R. 834, in a case similar to *Summers* and cited *Lewis* favorably as recently as 1995. Hollis v. Birch, [1995] S.C.R. 634. Commentators are also supportive of the idea of alternative liability. See DAN B. DOBBS, THE LAW OF TORTS, § 175 (2000); 4 FOWLER V. HARPER, FLEMING JAMES, JR. & OSCAR S. GRAY, THE LAW OF TORTS § 20.2, at 102-103 (2d ed. 1986); W. PAGE KEETON ET AL., PROSSER AND KEETON ON TORTS § 41, at 271 (5th ed. 1984). Alternative liability is supported by law and economics commentators for its deterrence benefits and accepted by at least one corrective-justice advocate as a pragmatic response to evidentiary uncertainty. See JULES L. COLEMAN, RISKS AND WRONGS 396-397 (1992); WILLIAM M.

LANDES & RICHARD A. POSNER, THE ECONOMIC STRUCTURE OF TORT LAW 212 (1987). But see Richard W. Wright, *Causation in Tort Law*, 73 CALIF. L. REV. 1735, 1817-1818 (1985) (suggesting that as the number of defendants increases, alternative liability is more justified by criminal-law principles than tort law). Professor Wright's additional criticism that alternative liability "disadvantages plaintiffs as well as defendants" because it is not available when fewer than all defendants acted tortiously proves too much. Any rule that has limits to its application disadvantages those who cannot satisfy those limits. The question is whether the limits are justified, not whether they disadvantage one side or another.

Only two jurisdictions have rejected the concept of alternative liability since the Second Restatement. See Leuer v. Johnson, 450 N.W.2d 363 (Minn.Ct.App.1990); Senn v. Merrell-Dow Pharms., Inc., 751 P.2d 215 (Or.1988). The decision by the *Leuer* court is unpersuasive. The Minnesota Supreme Court had previously rejected the rule of Ybarra v. Spangard, 154 P.2d 687 (Cal.1944), which applied res ipsa loquitur to several health-care professionals, even though they were not all legally responsible for the negligence that caused the plaintiff-patient's injury during surgery. The *Leuer* court predicted that the Minnesota Supreme Court would not adopt *Summers* "because [it] had as its basis *Ybarra*." Id. at 365. But *Summers* has roots in a far longer lineage of cases than *Ybarra*; indeed, the California Supreme Court in *Summers* cited Oliver v. Miles, 110 So. 666 (Miss.1927), a case factually quite similar to *Summers*. The rationale of alternative liability, imposing the risk of error on culpable defendants rather than an innocent plaintiff, is inapplicable to the *Ybarra* case. *Ybarra* sought to create an incentive for a tightly knit professional group who worked together to come forward with evidence that likely was possessed by many in the group. And, the Minnesota Supreme Court had earlier adopted § 433B(2), a closely analogous modification of the burden of proof, see Comment *d*, justifying it on *Summers* grounds and citing *Summers* in support of its decision. Mathews v. Mills, 178 N.W.2d 841 (Minn.1970). Particularly after the California Supreme Court rejected better access to evidence as a requirement for alternative liability in Sindell, the reasoning of the *Leuer* court is suspect. See also DAN B. DOBBS, THE LAW OF TORTS § 175, at 430 (2000) (courts could "reject *Ybarra* out of hand and still quite logically approve of *Summers*").

In *Senn*, the court proffered three grounds for rejecting alternative liability: 1) the large number of defendants involved; 2) the availability of modern discovery; and 3) that the rationale for alternative liability--culpable defendants and innocent plaintiff--was unrelated to the availability of causation evidence. The question of multiple defendants is addressed in Comment *j* and its Reporters' Note. As to the last ground, while the comparative culpability of the parties does not bear on access to evidence of causation, it does provide a rationale for reallocating the risk of nonproduction and nonpersuasion when evidence of an essential issue is unavailable. Finally, with regard to the discovery ground, in some cases the costs of obtaining evidence may be economically prohibitive; moreover, there are some kinds of evidence (e.g., nondocumentary evidence possessed by an adversary of that party) that can be very difficult, if not impossible, to obtain, even with modern discovery techniques.

In Spencer v. Baxter International, Inc., 163 F. Supp. 2d 74 (D.Mass.2001), plaintiff's decedent became infected with the HIV virus, likely as a result of contamination in a blood factor she received for hemophilia. The court concluded that, even if Massachusetts had adopted alternative liability, it would be inapplicable in that case because plaintiff had not joined as defendants all providers of the blood factor. The court added in its concluding paragraph that it believed the Massachusetts Supreme Judicial Court would not adopt alternative liability under the

circumstances. The only circumstance cited by the court was the plaintiff's failure to satisfy the requirements of alternative liability, which is not a reason for refusing to recognize alternative liability generally. Perhaps the court meant only that it did not believe the Massachusetts court would adopt alternative liability on the facts of the case before the district court.

Comment f. Rationale. For courts that have not required that defendants have superior access to evidence of which defendant was the cause of plaintiff's harm, see Sindell v. Abbott Laboratories, 607 P.2d 924, 930 (Cal.1980); Abel v. Eli Lilly & Co., 343 N.W.2d 164, 174 (Mich.1984). Indeed, as the *Sindell* court observed, even in Summers v. Tice, the defendants had no better idea of the cause of plaintiff's harm than did the plaintiff. *Sindell* at 929. Most courts do not even mention this matter in adopting or applying alternative liability.

A number of cases display concern with plaintiffs who failed diligently to pursue available avenues to identify the person who caused the harm, although in all instances that opportunity had passed. See Kinnett v. Mass. Gas & Elec. Supply Co., 716 F. Supp. 695 (D. N.H.1989) (applying Maine law); Long v. Krueger, Inc., 686 F. Supp. 514, 517, 519 (E.D.Pa.1988); Layton v. Blue Giant Equip. Co., 599 F. Supp. 93 (E.D.Pa.1984) (plaintiff, who should have been able to identify defendant if exercising reasonable diligence, denied alternative liability); Klein v. Council of Chem. Ass'ns, 587 F. Supp. 213, 222 (E.D.Pa.1984) ("[Plaintiff] was in a position to know the products used in his work environment."); Abel v. Eli Lilly & Co., 343 N.W.2d 164, 173 (Mich.1984) (requiring that "the plaintiffs, through no fault of their own, are unable to identify which actor caused the harm," as a condition for its modified alternative liability applicable to DES cases); Bixler v. Avondale Mills, 405 N.W.2d 428, 431 (Minn.Ct.App.1987); Cousineau v. Ford Motor Co., 363 N.W.2d 721, 727 (Mich.Ct.App.1985); cf. Bradley v. Firestone Tire & Rubber Co., 590 F. Supp. 1177 (D. S.D.1984) (tire rim manufactured with distinctive emblem that would permit identification if plaintiff's employer's worker's-compensation carrier had not lost it); Cummins v. Firestone Tire & Rubber Co., 495 A.2d 963 (Pa.Super.Ct.1985) (inability to identify defendant who caused plaintiff's harm was the result of third-party defendant's action in disposing of the product after the accident and therefore was not attributable to defendant manufacturers). This treatment is comparable to that accorded in cases involving product malfunctions in which the product becomes unavailable for examination after the accident. See Gary T. Schwartz, *New Products, Old Products, Evolving Law, Retroactive Law*, 58 N.Y.U. L. REV. 796, 835-836 (1983).

One court made the connection between the rationale for alternative liability with multiple defendants and the single-defendant case in which the plaintiff had difficulty proving causation based on a failure to warn her physician of the dangers of breast-implant failure. The court responded by adopting a presumption of factual causation in such failure-to-warn cases. See Hollis v. Birch, [1995] S.C.R. 634.

Comment g. Joinder of all defendants. Beginning in the 1980s, a number of plaintiffs sought to invoke alternative liability when they were unable to prove which defendant produced and sold the drug DES that their mother had taken a generation earlier when the plaintiff was in utero. Since then, plaintiffs with difficulties proving the manufacturers of asbestos to which they were exposed have also sought to invoke alternative liability to shift the burden of proof to defendants. The near-universal response has been to deny these efforts, in DES cases because plaintiffs did not join all of the manufacturers of DES who may have provided the drug taken by their mothers and in at least some asbestos cases for similar reasons. While some courts have fashioned a different burden-shifting device, market-share liability, alternative liability has been limited to

instances in which plaintiff has joined as defendants all the persons who may have caused the harm. In cases outside the DES and asbestos realm, as well, courts have refused to relax the requirement that plaintiff join all responsible parties.

In Doe v. Baxter Healthcare Corp., 380 F.3d 399 (8th Cir.2004) (applying Iowa law), the court stringently applied the rule requiring joinder of all defendants and held that a "very low" risk that a source other than the joined defendants provided the blood derivative that infected plaintiff with HIV precluded use of alternative liability against the defendants who provided plaintiff with Factor VIII derived from pooled blood. For other cases rejecting alternative liability in DES cases because of the failure to join all manufacturers, see, e.g., Sindell v. Abbott Labs., 607 P.2d 924, 931 (Cal.1980); Mulcahy v. Eli Lilly & Co., 386 N.W.2d 67, 73-74 (Iowa 1986); Zafft v. Eli Lilly & Co., 676 S.W.2d 241, 244 (Mo.1984); Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069 (N.Y.1989); Martin v. Abbott Labs., 689 P.2d 368, 595 (Wash.1984); Collins v. Eli Lilly Co., 342 N.W.2d 37, 46 (Wis.1984). Abel v. Eli Lilly & Co., 343 N.W.2d 164 (Mich.1984), is sometimes cited as an exception, a case permitting a DES plaintiff to proceed on an alternative-liability theory. But in *Abel*, the court recognized that the alternative-liability scheme it was adopting was considerably different from the existing alternative-liability rule: "we are actually fashioning and approving a new DES-unique version of alternative liability." Id. at 173. Notably, the *Abel* court required plaintiff to "bring before the court all the actors who may have caused the injury in fact." Id. For non-DES cases requiring that all tortfeasors be joined, see City of Philadelphia v. Lead Indus. Ass'n, 994 F.2d 112, 128 (3d Cir.1993) (applying Pennsylvania law) (lead-paint manufacturers); Sanderson v. Int'l Flavors & Fragrances, Inc., 950 F. Supp. 981, 989 (C.D.Cal.1996) (fragrance-product manufacturers); Barron v. Martin-Marietta Corp., 868 F. Supp. 1203, 1209 (N.D.Cal.1994) (manufacturers of canisters for surface-to-air missiles); Univ. Sys. v. U.S. Gypsum Co., 756 F. Supp. 640 (D.N.H.1991) (asbestos-products manufacturers); Long v. Krueger, Inc., 686 F. Supp. 514 (E.D.Pa.1988) (stool manufacturers); Marshall v. Celotex Corp., 651 F. Supp. 389 (E.D. Mich.1987) (asbestos-products manufacturers); Vigiolto v. Johns-Manville Corp., 643 F. Supp. 1454 (W.D.Pa.1986) (asbestos-products manufacturers); Black v. Abex Corp., 603 N.W.2d 182 (N.D.1999) (asbestos-containing brake and clutch products); Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691 (Ohio 1987) (asbestos-products manufacturers); Case v. Fibreboard Corp., 743 P.2d 1062 (Okla.1987) (asbestos-products manufacturers); Gaulding v. Celotex Corp., 772 S.W.2d 66 (Tex.1989) (asbestos-products manufacturers).

An oft-cited exception is Menne v. The Celotex Corp., 861 F.2d 1453 (10th Cir.1988), in which the court affirmed the use of alternative liability in an asbestos case where the plaintiff had not joined all possible asbestos-product manufacturers who contributed to his harm. *Menne*, however, is a narrower exception than is often recognized. First, the plaintiff was not attempting to shift the burden of proof to all (or a subset of) asbestos-product manufacturers generally; plaintiff introduced sufficient evidence to permit the jury to find exposure to the asbestos products of each of the defendants, although the duration and intensity of the exposure to each was unknown. Id. at 1461. Only because the *Menne* court found that Nebraska law would not be satisfied unless each of the asbestos defendants was a but-for cause (and because the court failed to appreciate that even if the plaintiff was exposed to a dose greater than that necessary to cause mesothelioma, the overdetermined-cause rule, see § 27, would apply to each of the defendants to whose asbestos products the plaintiff was exposed) was the court impelled to invoke a burden-shifting doctrine for causation. Since courts routinely permit the jury to draw an inference of

causation if sufficient exposure to a defendant's asbestos products is shown, the incremental effect of the *Menne* decision is only to impose the burden of persuasion on asbestos defendants. And, of course, plaintiff, if he is not relying on alternative liability, need not sue all persons who were a cause of the harm. *Menne* also joins a minority of courts that have not imposed a qualitative threshold of exposure to a defendant's asbestos products as an aspect of the plaintiff's burden of production.

While the ground for rejecting alternative liability in DES cases was the failure to join as defendants all of the potentially responsible manufacturers, that fact does not explain the undesirability of apportioning liability based on comparative responsibility. The existence of many plaintiffs harmed by a fungible product produced by many manufacturers calls for apportionment that more closely tracks the harm caused by each manufacturer, rather than apportionment based on culpability. With single-event and single-plaintiff torts, such as in Summers v. Tice, 199 P.2d 1 (Cal.1948), only one defendant caused all of the harm produced by the defendants' tortious acts, and therefore no need exists to employ a more causally related apportionment scheme. That is not the case in the multiple-parties situation, where the amount of harm caused by defendants is quite variable and may bear little or no relationship to the relative culpability among the defendants. For courts that have expressed concern about joint and several liability and the desirability of limiting defendants' liability to the amount of harm caused by each as grounds for rejecting alternative liability in multi-plaintiff products-liability cases, see City of Philadelphia v. Lead Indus. Ass'n, 904 F.2d 112, 128 (3d Cir.1993) (applying Pennsylvania law); Doe v. Cutter Biological, 852 F. Supp. 909, 916-917 (D. Idaho 1994); Conley v. Boyle Drug Co., 570 So. 2d 275, 285 (Fla.1999); Smith v. Cutter Biological, Inc., 823 P.2d 717 (Haw.1991); George v. Parke-Davis, 733 P.2d 507, 513 (Wash.1987); Collins v. Eli Lilly Co., 342 N.W.2d 37, 46 (Wis.1984); see also Richard W. Wright, *Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts*, 73 IOWA L. REV. 1001, 1075 (1988).

Settlement with some of the persons who may have caused plaintiff's harm is not inconsistent with the requirements and rationale of this Section. When settlement occurs with one or more of the persons who may have caused plaintiff's harm, there is no risk that the person who actually caused the plaintiff's harm will escape liability. Furthermore, the credit to nonsettling defendants for the loss of their contribution claim provides a reasonable method of apportioning liability among those held liable for the plaintiff's harm. See Gard v. Raymark Indus., Inc., 229 Cal. Rptr. 861, 870 (Ct.App.1986); Vahey v. Sacia, 178 Cal. Rptr. 559 (Ct.App.1981). But see Battocchi v. Wash. Hosp. Ctr., 581 A.2d 759, 771 (D.C.Ct.App.1990) (alternative ground for affirming denial of alternative-liability instruction was the absence of one possible tortfeasor due to pretrial settlement because the nonparty might "in attempting to exculpate himself ... have instead persuaded the jury that he alone was negligent or the sole cause of the injury"). The Restatement Second of Torts, in Comment *h* to § 433B(3), recognized that some adjustment to the joinder requirement for alternative liability might be necessary when "one of the actors ... cannot be joined as a defendant."

Because of the requirement of joining all potential tortfeasors and because this subsection shifts the burden of proof when two or more defendants' tortious conduct exposed the plaintiff to risk, this Section is unavailable when plaintiff sues only one defendant. See Lee v. Baxter Healthcare Corp., 721 F. Supp. 89 (D.Md.1989); cf. Rogers v. AAA Wire Prods., Inc., 513 N.W.2d 643 (Wis.Ct.App.1994).

Comment h. Each defendant acted tortiously. Cases continuing to reject application of alternative liability when the plaintiff fails to prove that all defendants acted tortiously, include Transco Leasing Corp. v. United States, 896 F.2d 1435, 1446 (5th Cir.1990) (Federal Tort Claims Act case in which Texas provided the applicable law); Burton v. Waller, 502 F.2d 1261 (5th Cir.1974) (applying Mississippi law in federally created civil-rights suit); Bradley v. Firestone Tire & Rubber Co., 597 F. Supp. 1177 (D.S.D.1984); Thodos v. Bland, 542 A.2d 1307 (Md.Ct.Spec.App.1988); Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691 (Ohio 1987); Tirey v. Firestone Tire & Rubber Co., 513 N.E.2d 825 (Ohio Ct. Common Pleas 1986); Clift v. Nelson, 608 P.2d 647 (Wash.Ct.App.1980). The Illustration is based (loosely) on Larson v. St. Francis Hotel, 188 P.2d 513 (Cal.1948).

Comment i. Exposing plaintiff to the risk of harm. Although often other grounds for denying alternative liability exist, a number of courts have invoked the concern that there be some reasonable "proximity" between the defendants' tortious conduct and the plaintiff's harm. Thus, in virtually all products-liability cases, unless the plaintiff can show exposure to the product, at a minimum, courts refuse to apply alternative liability. See Vigiolto v. Johns-Manville Corp., 643 F. Supp. 1454 (W.D.Pa.1986), aff'd, 826 F.2d 1058 (3d Cir.1987); Klein v. Council of Chem. Ass'ns, 587 F. Supp. 213, 222 (E.D.Pa.1984) (plaintiff unable to identify the chemical products or manufacturers to which he had been exposed and which allegedly caused his bladder cancer); Setliff v. E.I. DuPont de Nemours & Co., 38 Cal. Rptr. 2d 763 (Ct.App.1995); Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691, 696-697, 698 (Ohio 1987) (equating the requirement that plaintiff show that all defendants acted tortiously with the plaintiff showing that defendants supplied asbestos products to plaintiff's place of employment); Cummins v. Firestone Tire & Rubber Co., 495 A.2d 963 (Pa.Super.Ct.1985). The Fifth Circuit attempted to capture this concern in an asbestos case, in which the plaintiff could not establish exposure to the products of four defendants and sought to rely on several alternative-liability cases: "All of these concern defendants who were proved to have some factual connection with the plaintiff's injury; to apply them to defendants as to which there is no proof of any such connection would beg the question of causation entirely." Thompson v. Johns-Manville Sale Corp., 714 F.2d 581, 582-583 (5th Cir.1983) (applying Louisiana law); see also Kinnett v. Mass. Gas & Elec. Supply Co., 716 F. Supp. 695 (D.N.H.1989); Shackil v. Lederle Labs., 561 A.2d 511, 520 (N.J.1989) (requiring "some 'reasonable connection' be established between defendant and the ultimate harm"); Case v. Fibreboard Corp., 743 P.2d 1062 (Okla.1987); cf. Abel v. Eli Lilly & Co., 343 N.W.2d 164, 172 (Mich.1984) (distinguishing *Summers* from the DES cases: "in *Summers*, each defendant was negligent toward *the* plaintiff; here, each defendant was negligent toward *a* plaintiff, but each defendant was not negligent toward each plaintiff," but nevertheless crafting a modified-for-DES alternative-liability doctrine). But see In re Agent Orange Prod. Liab. Litig., 597 F. Supp. 740, 822 (E.D.N.Y.1984) (class-action settlement, decision on fairness of settlement); Wysocki v. Reed, 583 N.E.2d 1139 (Ill.App.Ct.1991) (legal-malpractice action against plaintiff's former counsel; court concluded that alternative-liability theory would have been available in action on plaintiff's behalf against 2 manufacturers of drug that caused adverse reaction).

A few courts have rejected alternative liability when the defendants did not expose the plaintiff to comparable risks or fungible products. In almost all such cases, the product was asbestos, and there were other and better reasons for rejecting the application of alternative liability. See, e.g., Rutherford v. Owens-Ill., Inc., 941 P.2d 1203 (Cal.1997); Horton v. Harwick Chem. Corp., 653 N.E.2d 1196 (Ohio 1995); Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691 (Ohio

1987). The rejection of market-share liability in asbestos litigation because asbestos products are not fungible and do not pose similar risks, see Comment *o*, is probably why these courts adopted this ground for rejecting alternative liability. As explained in Comment *o*, there are good reasons in the market-share context for insisting on fungible products and comparable risks. Outside of asbestos litigation, courts have not articulated such a requirement for alternative liability, although in the vast majority of cases in which alternative liability is involved, the risks imposed by defendants on plaintiff are comparable or nearly so. In cases with two defendants and noncomparable risks, plaintiffs may not need the burden shifting provided by alternative liability because the difference in risk may enable the plaintiff to prove by a preponderance of the evidence that one defendant was the cause of the harm. For cases in which the risks were not comparable (or apparently not comparable) and courts nevertheless affirmed the use of alternative liability, see Bowman v. Redding & Co., 449 F.2d 956 (D.C. Cir.1971); Hood v. Hagler, 606 P.2d 548 (Okla.1979) (although both defendant dog owners permitted their dogs to run loose, no evidence was presented that the dogs were equally prone to bite someone without provocation).

Comment j. Multiple actors and nonsimultaneous tortious conduct. Restatement Second, Torts § 433B(3) made alternative liability available regardless of the number of defendants, and courts continue to adhere to that rule. The most notable exception is Senn v. Merrell-Dow Pharms., Inc., 751 P.2d 215 (Ore.1988), a DES case. The *Senn* court reviewed a number of DES cases in which alternative liability had been rejected. The court then synthesized alternative liability and the cases it surveyed by observing,

> [M]ost courts considering the issue seem to feel that a rule of alternative liability modeled on *Summers* and Section 433B is defensible where the number of defendants is small, but that such a rule breaks down and becomes "unfair" at some undefined point as the number of defendants increases and the likelihood that any particular defendant actually caused plaintiff's harm decreases.

Id. at 222; see also DAN B. DOBBS, THE LAW OF TORTS § 175, at 428-429 n.15 (2000) (suggesting an implicit view by some courts that with 2 defendants the probability is very close to what is ordinarily required for adequate proof, but that as the number of defendants increases, the probability recedes from that close point). The court raised another concern about multiple defendants, alternative liability, and burden shifting: in a case with only two defendants and presumably a facial 50 percent probability of each having caused plaintiff's harm, even a scintilla of exonerative evidence by a defendant would permit the jury to find that defendant not liable; but in a case with three or more defendants, "what proof would be sufficient to satisfy the defendant's burden of proof?," the court inquired rhetorically.

However, none of the cases cited by the *Senn* court rejected alternative liability in DES cases because of the number of defendants. Most were concerned that plaintiffs had not joined all the manufacturers of DES. A few mentioned that alternative liability did not provide an attractive means for apportioning liability among defendants and therefore crafted another system to circumvent the burden of proof. And while the likelihood that any defendant caused plaintiff's injury does decrease as the number of defendants increases, so does the proportion of damages to be paid by each defendant. Defendants would be able to satisfy their burden of production when three or more defendants are subject to alternative liability in one of two ways: a defendant might show why it was not the cause of plaintiff's injury or it might show which one of the other defendants was the cause. While in many cases defendants may be unable to submit such

evidence, there is no conceptual difficulty with exoneration in alternative-liability cases with more than two defendants. Interestingly, the objections of the *Senn* court to alternative liability with more than two defendants were not presented on the facts of that case, which involved only two manufacturers of a vaccine. Although the situation does not occur very often, courts have employed alternative liability when there are more than two defendants, and no court has refused to apply alternative liability solely because the number of defendants was greater than two. See Doe v. Baxter Healthcare Corp., 380 F.3d 399 (8th Cir.2004) (applying Iowa law) (3 defendants who provided Factor VIII; court holds that alternative liability is not available but does not suggest that number of defendants is a ground for inapplicability); Zands v. Nelson, 797 F. Supp. 805 (S.D.Cal.1992) (federal law under Resource Conservation and Recovery Act; 3 defendants); Poole v. Alpha Therapeutic Corp., 696 F. Supp. 351 (N.D.Ill.1988) (all manufacturers of Factor VIII blood product); Vahey v. Sacia, 178 Cal. Rptr. 559, 564 (Ct.App.1981) (4 possible tortfeasors); McCoy v. DeLiefde, 135 N.W.2d 916 (Mich.1965) (3 hunters); NOPCO Chem. Div. v. Blaw-Knox Co., 281 A.2d 793 (N.J.1971) (4 defendants); McMillan v. Mahoney, 393 S.E.2d 298 (N.C.Ct.App.1990) (6 defendants); Snoparsky v. Baer, 266 A.2d 707 (Pa.1970) (12 defendants). At least one case prior to the Restatement Second of Torts accepted alternative liability with three defendants. Benson v. Ross, 106 N.W. 1120 (Mich.1906). European law is generally in accord, permitting alternative liability to be invoked when more than two defendants are involved. See EUROPEAN GROUP ON TORT LAW, PRINCIPLES OF EUROPEAN TORT LAW Art. 3:103(1), at http:// civil.udg.es/tort/Principles/text.htm (last updated Feb. 9, 2005) ("In case of multiple activities, where each of them alone would have been sufficient to cause the damage, but it remains uncertain which one in fact caused it, each activity is regarded as a cause to the extent corresponding to the likelihood that it may have caused the victim's damage.").

A number of courts, some prompted by Comment *h* to § 433B(3) of the Restatement Second of Torts, have adopted the rule, or stated, that simultaneous tortious conduct by the defendants is required. Comment *h* did not impose a simultaneity requirement. Indeed, it did not impose any requirements, but merely observed that in all cases to that time the tortious conduct had occurred at the same time or nearly so, as well as identifying several other common characteristics of those cases. The black letter of § 433B(3) mentions no simultaneity requirement. Comment *h* contained an invitation to relax the requirements of the rule stated in § 433B(3). In a puzzling sentence that invites courts to relax the requirements of the rule in future cases, the Comment mentions not only joinder of all defendants (a requirement of the rule) but also two other factors that are not made requirements of § 433B(3), but, like simultaneity, had been common characteristics of alternative-liability cases:

> It is possible that cases may arise in which some modification of the rule stated may be necessary because of complications arising from the fact that one of the actors involved is not or cannot be joined as a defendant, or because of the effect of lapse of time, or because of substantial differences in the character of the conduct of the actors or the risks which they have created.

That sentence omits simultaneity in its invitation to relaxation, inviting conflicting, but unsatisfying, interpretations: since simultaneity is not a requirement, it need not be relaxed; or simultaneity should not be relaxed. The difficulty with the first interpretation is that the sentence includes two other nonrequirement circumstances for its invitation to relaxation. The difficulty with the second interpretation is that there is no reason to single out simultaneity for different

treatment from the requirement and other circumstances listed in the sentence. Perhaps the best explanation is that this omission was a casual oversight. Most importantly, the Restatement Second of Torts identifies no reason why simultaneity should be required. Nor do the cases that have invoked it. And in virtually every one, other and more persuasive grounds existed for denying application of alternative liability.

The first case to invoke a simultaneity requirement, Starling v. Seaboard Coast Line Railroad, 533 F. Supp. 183, 191 (S.D.Ga.1982), an asbestos case, simply identified it as a requirement (along with joinder of all tortfeasors) and cited *Sindell* in support. *Sindell* imposed no such requirement for alternative liability, rejecting its application in that DES case because plaintiff had joined only five of several hundred DES manufacturers. *Sindell,* 607 P.2d at 928-931. Although *Starling* does not explain which asbestos defendants were sued, a more persuasive reason for rejecting alternative liability appears to be the failure to join all tortious actors or the failure to identify those to whose asbestos products plaintiff was exposed. See Comments *i* and *k*.

In another case invoking simultaneity, Smith v. Cutter Biological, Inc., 823 P.2d 717 (Haw.1991), the court cited *Starling* and provided no explanation for such a requirement. The case involved market-share concerns, with persons harmed by multiple manufacturers of an alleged defective product but no evidence about which manufacturer's product harmed which person. The court sought to avoid imposing joint and several liability and to limit each defendant's liability to the total amount of harm caused by each. With an unmodified alternative-liability theory, that outcome was not possible, as alternative liability, when invoked, results in joint and several liability. See Comment *g*. Another case that invoked a lack of simultaneity as a ground for denying alternative liability but that did not explain the purpose served by the requirement, and in which there were other persuasive reasons for rejecting alternative liability, is Santarelli v. BP America, 913 F. Supp. 324 (M.D.Pa.1996) (suit against multiple wholesalers for contaminated food; no evidence that each of wholesalers was selling contaminated food and hence had acted tortiously).

In City of Philadelphia v. Lead Industries Association, Inc., 904 F.2d 112 (3d Cir.1993) (applying Pennsylvania law), a suit against lead-pigment manufacturers and their trade association, the court addressed defendants' argument that the Pennsylvania Supreme Court had only approved alternative liability when the defendants' tortious conduct was simultaneous. That fact existed in Snoparsky v. Baer, 266 A.2d 707 (Pa.1970), in which the court approved alternative liability, but the fact went unremarked upon by the court. The Third Circuit also confronted the claim that all potential tortfeasors had to be joined before alternative liability could be employed. The court proceeded to conclude that the modifications sought by plaintiffs would transform alternative liability into market-share liability and objected that this transformation would produce inequitable apportionment among defendants. See Comment *g*. But that inequitable apportionment would be solely the result of the failure to join all potential tortfeasors; simultaneity is irrelevant to that concern. Thus, *City of Philadelphia* provides no good reason for a simultaneity requirement.

Skipworth v. Lead Industries Association Inc., 690 A.2d 169 (Pa.1997), characterized *Snoparsky* as requiring simultaneous conduct and concluded that because defendants manufactured lead pigment over a 100-year period with some moving in and out of the industry during that time, alternative liability was unavailable. The concern that some of the defendants could not have been responsible for plaintiff's harm because they did not manufacture lead pigment when it could have been in paint used in plaintiff's home is addressed by the requirement that each

defendant's tortious act expose the plaintiff to the risk of harm. See Comment *i*. Simultaneity is a redundant and overly broad restriction to protect this concern. The failure of the plaintiff to join all lead-pigment manufacturers was an independent and sufficient ground for denying alternative liability in *Skipworth*.

In summary, simultaneity is both an unnecessary and unsupportable requirement: in each of the cases invoking it, there were other adequate grounds for rejecting alternative liability; no cogent purpose has been articulated for its retention. See also DAN B. DOBBS, THE LAW OF TORTS § 175, at 429 (2000) (characterizing simultaneity as an example of a limitation that "seem[s] to have no essential connection with [the rule]").

For cases that have affirmed the use of alternative liability, despite an absence (or apparent absence) of simultaneity, see Poole v. Alpha Therapeutic Corp., 696 F. Supp. 351, 356 n.3 (N.D.Ill.1988) (rejecting defendant's claim that simultaneity is required for alternative liability); Wysocki v. Reed, 583 N.E.2d 1139 (Ill.App.Ct.1991) (concluding that alternative liability would be available against 2 drug manufacturers for failure to warn in a legal-malpractice case against the plaintiff's former counsel); Jackson v. Glidden Co., 647 N.E.2d 879 (Ohio Ct.App.1994) (reversing trial court's dismissal on the pleadings); cf. In re Agent Orange Prod. Liab. Litig., 597 F. Supp. 740, 822 (E.D.N.Y.1984); NOPCO Chem. Div. v. Blaw-Knox Co., 281 A.2d 793 (N.J.1971) (consecutive bailees of commercial machine damaged while it was in the possession of one of the bailees); Minnich v. Ashland Oil Co., 473 N.E.2d 1199 (Ohio 1984) (plaintiff alleged defendants failed to provide adequate warnings with chemicals provided to plaintiff's employer). At least one case prior to the Restatement Second did not require simultaneity. Benson v. Ross, 106 N.W. 1120 (Mich.1906) (3 defendants, each shooting the same gun serially).

Comment k. One or more defendants' tortious conduct caused plaintiff's harm. See Daniels v. Smith, 471 S.W.2d 508 (Mo.Ct.App.1971) (plaintiff, who claimed that the negligence of 3 automobile drivers caused her harm, could not invoke alternative liability); James v. Chevron U.S.A., Inc., 694 A.2d 270, 283 (N.J.Super.Ct.App.Div.1997), aff'd, 714 A.2d 898 (N.J.1998) (plaintiff's decedent exposed to chemicals of multiple defendants; alternative liability inapplicable because of allegations that exposure to all chemicals caused death).

Illustration 4 reveals the interaction between Comment *d*, which addresses the situation in which two or more tortfeasors each contribute to the plaintiff's harm, and Subsection (b), in which only one tortfeasor causes plaintiff's harm. Because of uncertainty about whether only one defendant caused plaintiff's harm or both contributed to the harm, the jury must make preliminary findings to determine which of the two rules is applicable. The difference between the two rules in the outcome of the case is that, if Comment *d* is applicable, causal apportionment is the applicable method for apportioning liability among the defendants provided there is sufficient evidence to do so. See Restatement Third, Torts: Apportionment of Liability § 26, Comment *h*. Comment *d* and Subsection (b) are closely related both in presenting similar problems of proof and employing common rationales to modify the burden of proof. See W. PAGE KEETON ET AL., PROSSER AND KEETON ON TORTS § 41, at 271 n.60 (5th ed. 1984) (describing alternative liability as merely an extension of the rule provided in Comment *d*).

Comment l. Exposure to multiple defendants' toxic products. Courts have repeatedly declined to permit plaintiffs who could not identify the manufacturer of the product causing them harm to employ alternative liability to shift the burden of proof to defendants on causation. Even in those relatively rare instances in which all of the products on the market were defective, thereby

satisfying the requirement that all defendants acted tortiously, the transaction costs of a contrary rule are too substantial to justify casting the alternative-liability rule this broadly. See Comment *h*. When plaintiff does prove exposure to multiple defendants' products and the products and harm are consistent with increased risk due to increased exposure, as is the case with asbestos, courts have held that all defendants may be held liable for the plaintiff's harm, see § 27, Comment *g*, thereby both obviating the need for a plaintiff to invoke alternative liability and making it inapplicable. See Comment *i*; Rutherford v. Owens-Ill., Inc., 941 P.2d 1203, 1218-1220 (Cal.1997) (explaining that asbestos plaintiffs are not required to prove *the* specific exposure or dose that reached the causal threshold); Celotex Corp. v. Tate, 797 S.W.2d 197, 204 (Tex.Ct.App.1990).

Comment m. Procedural implications. For an example of a jury instruction invoking the burden shifting provided by this subsection, see BAJI No. 3.80 (1999 WL 117693, at *41) (imposing both burden of production and persuasion on defendants). Illustration 11 is based very loosely on Bowman v. Redding & Co., 449 F.2d 956 (D.C.Cir.1971), which also addresses procedural aspects of the invocation of alternative liability. In Illustration 13, because resolution of the credibility of Alan, Barbara, and Chad is a matter for the jury, Penelope has satisfied her burden of production of negligence by each of them. If the jury accepts Chad's testimony, he is not negligent, and either Alan or Barbara's negligence in continuing to fire their paintball guns after Chad sounded the warning was a cause of Penelope's harm, thereby making alternative liability appropriate for the two of them. If the jury accepts Alan and Barbara's testimony and rejects Chad's, his negligence in not sounding the warning is the cause of Penelope's harm, and alternative liability is unnecessary.

Comment n. Plaintiff negligence. Despite several decades of comparative responsibility, the Reporters have been able to identify only two cases in which the suggestion of plaintiff negligence existed along with a claim based on alternative liability. One such case is Bowman v. Redding & Co., 449 F.2d 956 (D.C.Cir.1971). The court did not face the question of reconciling alternative liability and plaintiff negligence because the District of Columbia is a jurisdiction in which contributory negligence remains a complete bar to recovery. The second such case is Vahey v. Sacia, 178 Cal. Rptr. 559 (Ct.App.1981). In that case, the court reversed a judgment for defendant based on the failure of the trial court to give an alternative-liability instruction. Defendant argued that the instruction was inappropriate because the plaintiff's failure to wear a seat belt may have also caused or contributed to her injuries. The court's initial response was that the jury should be instructed that only if it found the plaintiff was not negligent would alternative liability and a concomitant shift in the burden of proof be appropriate, which is consistent with the suggestion in this Comment. However, the court went on to suggest that comparative responsibility was not inconsistent with the rationale for alternative liability but failed to articulate that rationale. Id. at 563. Later in its opinion, the court, in responding to another argument, stated the basis of alternative liability to be the defendant's acting in a way that deprived the plaintiff of the ability to prove her claim. However, in the same sense that the defendant's (and other tortfeasors') negligence in causing the plaintiff harm created the difficulty of proof, so did the plaintiff's contributory negligence, without which the plaintiff would not have been harmed and therefore not have faced the difficulty of proving which defendant's negligence was a cause of the harm.

Professor Dobbs observes that, when the plaintiff is also at fault, "the strong moral basis for treating the negligent defendants as causes in fact of the harm could become quite attenuated."

DAN B. DOBBS, THE LAW OF TORTS § 175, at 429 (2000).

Comment o. Market-share liability. The California Supreme Court was the first to adopt a market-share system for DES manufacturers. Sindell v. Abbott Labs., 607 P.2d 924 (Cal.1980). But the court required an additional eight years before filling in additional important details, and the question of what constitutes joinder of a substantial share of the market, a requirement of *Sindell*, still remains uncertain. See Brown v. Superior Court, 751 P.2d 470 (Cal.1988) (holding that market-share defendants are liable only for their market-share percentage of plaintiff's damages and are not jointly and severally liable); Murphy v. E.R. Squibb & Sons, Inc., 710 P.2d 247 (Cal.1985) (10% share of DES market did not constitute substantial share of the market for purposes of market-share theory). Other courts adopting a market-share theory in DES litigation include McCormack v. Abbott Labs., 617 F. Supp. 1521 (D.Mass.1985); McElhaney v. Eli Lilly & Co., 564 F. Supp. 265 (D.S.D.1983) (concluding that South Dakota Supreme Court would adopt alternative-liability theory in DES litigation); Abel v. Eli Lilly & Co., 418 Mich. 311, 343 N.W.2d 164 (Mich.1984); Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069 (N.Y.1989); Martin v. Abbott Labs., 689 P.2d 368 (Wash.1984); Collins v. Eli Lilly Co., 342 N.W.2d 37 (Wis.1984) (adopting a "risk-contribution" theory that apportions liability to DES manufacturers according to their comparative responsibility).

For courts that have declined to adopt a market-share liability scheme either for DES cases or in general, see Wood v. Eli Lilly & Co., 38 F.3d 510 (10th Cir.1994) (applying Oklahoma law); Tidler v. Eli Lilly & Co., 851 F.2d 418 (D.C.Cir.1988); Starling v. Seaboard Coast Line R.R. Co., 533 F. Supp. 183, 190 (S.D.Ga.1982) (suggesting that the appropriate institution to address and fashion remedies for asbestos victims unable to prove which manufacturers' product to which they were exposed is the legislature); Mizell v. Eli Lilly & Co., 526 F. Supp. 589 (D.S.C.1981); Gray v. United States , 445 F. Supp. 337 (S.D.Tex.1978); Smith v. Eli Lilly & Co., 560 N.E.2d 324 (Ill.1990); Mulcahy v. Eli Lilly & Co., 386 N.W.2d 67, 75 (Iowa 1986) (expressing concern that modifying causation requirement is more appropriately the domain of the legislature); Zafft v. Eli Lilly & Co., 676 S.W.2d 241 (Mo.1984); Sutowski v. Eli Lilly & Co., 696 N.E.2d 187 (Ohio 1998).

Courts that have sought a narrow definition of the relevant market include Conley v. Boyle Drug Co., 570 So. 2d 275, 283-284 (Fla.1990); George v. Parke-Davis, 733 P.2d 507, 512 (Wash.1987) (market should be as narrow as possible to impose liability on only those companies most likely to have produced drug that caused plaintiff's harm). Courts rejecting a local or narrower basis for determining market share because of the transaction costs involved in determining a market sharply focused on the specific plaintiff include Smith v. Cutter Biological, Inc., 823 P.2d 717, 728 (Haw.1991); Hymowitz v. Eli Lilly & Co., 539 N.E.2d 1069, 1077 (N.Y.1989). Professor Twerski reports that litigants in California reached this same outcome by agreement. See Aaron D. Twerski, *Market Share--A Tale of Two Centuries*, 55 BROOK. L. REV. 869, 870 n.8 (1989). Other courts expressing concern about the problems of proof of the relevant market and its concomitant costs include Smith v. Eli Lilly & Co., 560 N.E.2d 324, 337-338 (Ill.1990) (quoting from California trial judge's expressing frustration with the absence of market-share evidence); Collins v. Eli Lilly Co., 342 N.W.2d 37, 48-49 (Wis. 1984) ("We view defining the market and apportioning market share as a near impossible task if it is to be done fairly and accurately in order to approximate the probability that a defendant caused the plaintiff's injuries. Further, we conclude that the waste of judicial resources which would be inherent in a second 'mini-trial' to determine market share militates against its adoption by this court.").

DES is a pathognomomic (or signature) agent; it is virtually the only cause of vaginal adenocarcinoma, the most serious consequence of in utero DES exposure in young women. That means that there is almost no uncertainty about agent-disease causation, even if there is a lack of proof of which *manufacturer* sold the agent that caused a plaintiff's disease. Most of the courts adopting market-share liability theories mention this fact when explaining the background of plaintiff's suit but do not rely on it as important in adopting market share. Most courts refusing to adopt market-share liability have not dwelt on this aspect, especially in asbestos litigation, where other quite significant causes of lung cancer do exist. However, a handful of courts emphasize this distinction between DES and other substances such as asbestos and lead paint in deciding that market-share liability should not be used in the latter situations. See Santiago v. Sherwin-Williams Co., 782 F. Supp. 186, 192-193 (D.Mass.1992); Starling v. Seaboard Coast Line R.R. Co., 533 F. Supp. 183, 190-191 (S.D.Ga.1982); Brenner v. Am. Cyanamid Co., 699 N.Y.S.2d 848 (App.Div.1999); Case v. Fibreboard Corp., 743 P.2d 1062, 1066 (Okla.1987).

For courts rejecting use of market-share liability in asbestos litigation on the grounds, inter alia, that asbestos products are not fungible and present different risks of harm, see Robertson v. Allied Signal, Inc., 914 F.2d 360 (3d Cir.1990) (applying Pennsylvania law); White v. Celotex Corp., 907 F.2d 104 (9th Cir.1990) (applying Arizona law); Blackston v. Shook & Fletcher Insulation Co., 764 F.2d 1480 (11th Cir.1985) (applying Georgia law); Starling v. Seaboard Coast Line R.R. Co., 533 F. Supp. 183, 190-191 (S.D.Ga.1982); Marshall v. Celotex Corp., 651 F. Supp. 389 (E.D. Mich.1987); Rutherford v. Owens-Ill., 941 P.2d 1203 (Cal.1997); Celotex Corp. v. Copeland, 471 So. 2d 533, 538 (Fla.1985); Leng v. Celotex Corp., 554 N.E.2d 468 (Ill.App.Ct.1990); Black v. Abex Corp., 603 N.W.2d 182 (N.D.1999); Goldman v. Johns-Manville Sales Corp., 514 N.E.2d 691, 700 (Ohio 1987); Case v. Fibreboard Corp., 743 P.2d 1062 (Okla.1987); see also Santiago v. Sherwin-Williams Co., 782 F. Supp. 186, 190 n.7 (D.Mass.1992) (citing cases); Brenner v. Am. Cyanamid Co., 699 N.Y.S.2d 848 (App.Div.1999) (rejecting market share in suit against manufacturers of lead carbonate, the lead ingredient in lead paint); Skipworth v. Lead Indus. Ass'n, Inc., 690 A.2d 169, 173 (Pa.1997) (rejecting market-share liability in lead-pigment litigation because of lack of fungibility). But see Wheeler v. Raybestos-Manhattan, 11 Cal. Rptr. 2d 109 (Ct.App.1992) (finding brake pads to which plaintiff was exposed to be fungible because they all contained the same type of asbestos and roughly the same weight of asbestos; the court did not consider the respective friability of the brake pads, which would affect their toxicity).

On the application of market share to manufacturing defects, compare Sheffield v. Eli Lilly & Co., 192 Cal. Rptr. 870 (Ct.App.1983) (market-share liability inapplicable to manufacturing defects) with Morris v. Parke, Davis & Co., 667 F. Supp. 1332 (C.D.Cal.1987) (market share applicable to manufacturing defects when all manufacturers in the industry share the process that produces the defects); Shackil v. Lederle Labs., 561 A.2d 511, 522 (N.J.1989) (concluding that one type of DPT vaccine may involve comparable risks from contamination with toxins but expressing doubt about another formulation of the vaccine).

For the limited cases in which a market-share theory of liability has been applied outside of DES, see Ray v. Cutter Labs., 754 F. Supp. 193 (M.D.Fla.1991) (factor VIII manufacturers); Morris v. Parke, Davis & Co., 667 F. Supp. 1332 (C.D.Cal.1987) (DPT vaccine manufacturers); In re Agent Orange Prod. Liab. Litig., 597 F. Supp. 740 (E.D.N.Y.1984) (Agent Orange manufacturers); Smith v. Cutter Biological, Inc., 823 P.2d 717 (Haw.1991) (factor VIII manufacturers); Jackson v. Glidden Co., 647 N.E.2d 879 (Ohio Ct.App.1994) (lead and/or lead-

paint manufacturers), overruled by Sutowski v. Eli Lilly & Co., 696 N.E.2d 187 (Ohio 1998). The vast majority of decisions outside DES reject market-share liability, often because of a lack of fungibility, but for a number of additional reasons as well. See, e.g., Brenner v. Am. Cyanamid Co., 699 N.Y.S.2d 848 (App.Div.1999); Hamilton v. Beretta U.S.A. Corp., 2001 WL 429247 (N.Y. Apr. 26, 2001); see also DAN B. DOBBS, THE LAW OF TORTS § 176, at 431-432 (2000) ("When it comes to other products besides DES, the theory has been invoked a few times, but even courts that accept market share theories for DES may reject the theory as inappropriate on the facts ..."); Aaron D. Twerski, *Market Share--A Tale of Two Centuries*, 55 BROOK. L. REV. 869, 875 (1989) (predicting, in the aftermath of *Hymowitz*, that market-share liability would not be widely employed).

Commentators are similarly split on the desirability of market-share liability; much academic effort has been devoted to the details of the different market-share theories adopted by the courts cited above. Compare David Fischer, *Products Liability--An Analysis of Market Share Liability*, 34 VAND. L. REV. 1623 (1981); James A. Henderson & Aaron D. Twerski, *Intuition and Technology in Product Design Litigation: An Essay in Proximate Causation*, 88 GEO. L.J. 659, 660 (2000) (market-share liability is an idea that just didn't work); Victor E. Schwartz & Liberty Mahshigian, *Failure to Identify the Defendant in Tort Law: Towards a Legislative Solution*, 73 CALIF. L. REV. 941 (1985) (urging that legislatures should be the engine for reform on this issue); Jonathan B. Newcomb, Comment, *Market Share Liability for Defective Products: An Ill-Advised Remedy for the Problem of Identification*, 76 NW. U. L. REV. 300 (1981); with Aaron D. Twerski, *Market Share--A Tale of Two Centuries*, 55 BROOKLYN L. REV. 869 (1989) (applauding *Hymowitz* as a logical solution to the DES-causation problem, but suggesting market share has limited applicability); Naomi Sheiner, Comment, *DES and a Proposed Theory of Enterprise Liability*, 46 FORDHAM L. REV. 963 (1978) (early article advocating an industry-liability theory that was quite influential in *Sindell*).

For the perspective of corrective-justice adherents on market-share theories, compare Ernest J. Weinrib, *Causation and Wrongdoing*, 63 CHI.-KENT L. REV. 407 (1987) (arguing that causation is essential to the corrective-justice mission of tort law by particularizing the plaintiff who has been harmed by the risk-creating conduct of a defendant) with Christopher H. Shroeder, *Corrective Justice and Liability for Increasing Risk*, 37 UCLA L. Rev. 429 (1990) (claiming that imposing liability for creating risk, even before harm has occurred, is consistent with corrective-justice principles) and Kenneth W. Simons, *Jules Coleman and Corrective Justice in Tort Law: A Critique and Reformulation*, 15 Harv. J.L. & Pub. Pol. 849, 884-885 (1992) (market-share liability can be squared with corrective justice by conceptualizing the harm as creating risk of injury and even can be squared with nonexculpation rule of *Hymowitz* on the ground it furthers accuracy in determining the harm caused by each defendant). Professor Richard Wright, a forceful corrective-justice scholar, has expressed comfort about the compatibility of corrective justice and market-share liability: "[I]f each defendant is held liable only for her share of the risk exposure [i.e., several liability for the market share of that defendant], there is no conflict with the corrective-justice view." Richard W. Wright, *Causation, Responsibility, Risk, Probability, Naked Statistics, and Proof: Pruning the Bramble Bush by Clarifying the Concepts*, 73 Iowa L. Rev. 1001, 1073 (1988); see also Richard W. Wright, *Once More into the Bramble Bush: Duty, Causal Contribution, and the Extent of Legal Responsibility*, 54 Vand. L. Rev. 1071, 1118 n.163 (2001) (characterizing market-share liability as second-best theory to imposing liability based on a factual causal relation between defendant's tortious conduct and plaintiff's harm).

For commentary on the application of market-share theories to specific products, see Andrew R. Klein, *Beyond DES: Rejecting the Application of Market Share Liability in Blood Products Litigation*, 68 TUL. L. REV. 883 (1994); Christina Bohannan, Note, *Product Liability: A Public Policy Approach to Contaminated Factor VII Blood Products*, 48 FLA. L. REV. 263 (1996); Shirley H. Fang, Comment, Santiago v. Sherwin-Williams Co.: *Rejection of Market Share Liability in Lead-Based Paint Litigation*, 43 BUFF. L. REV. 725 (1995); Kenneth R. Lepage, Note, *Lead-Based Paint Litigation and the Problem of Causation: Toward a Unified Theory of Market Share Liability*, 37 B.C. L. REV. 155 (1995).

Case Citations July 2005 -- November 2005:

S.D.N.Y.2005. Com. (o) cit. in ftn. (Prop. Final Draft No. 1, 2005). Several municipalities and water suppliers brought consolidated multidistrict action against gasoline producers, seeking relief from actual or possible groundwater contamination stemming from producers' use of the gasoline additive methyl tertiary butyl ether (MTBE). Denying several of defendants' motions to dismiss, the court examined several theories of collective liability, including market-share liability, under which liability was based on each defendant's share of the product's market, so that each defendant's liability would approximate the harm caused by that defendant's product. The court said that market-share liability was uniquely suited to cases involving fungible products, such as gasoline, because such products by nature were nonidentifiably interchangeable and posed equal risks of harm to those exposed to them. The court considered several factors in deciding whether to apply market-share liability, and then ruled that certain plaintiffs could use this theory against certain defendants. In re Methyl Tertiary Butyl Ether (MTBE) Products Liability Litigation, 379 F.Supp.2d 348, 376.

FN* This paragraph has been added since Tentative Draft No. 2 was approved by the membership in May 2002. It was discussed by the Council at its December 2004 meeting but not approved at that time. This paragraph, along with a Reporters' Note explaining it, was subsequently circulated to the Advisers for comment and was favorably received. It is presented to the membership for approval.

FNa As of the date of publication, this Draft has not been considered by the members of The American Law Institute and does not represent the position of the Institute on any of the issues with which it deals. The action, if any, taken by the members with respect to this Draft may be ascertained by consulting the Annual Proceedings of the Institute, which are published following each Annual Meeting.

(2005)

REST 3d TORTS-PH § 28

END OF DOCUMENT